```
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 08:47:04 ON 10 JUN 2004

=> file medline, biosis, embase
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 08:47:18 ON 10 JUN 2004

FILE 'BIOSIS' ENTERED AT 08:47:18 ON 10 JUN 2004
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FILE 'EMBASE' ENTERED AT 08:47:18 ON 10 JUN 2004
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=> s (AMPA adj receptor or AMPAR) AND treat?
L1          57 (AMPA ADJ RECEPTOR OR AMPAR) AND TREAT?

=> s l1 and clinical or animal or in adj vivo or mammal or mouse or mice or rat or
monkey or human or rabbit or animal
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
<------User Break------>

SEARCH ENDED BY USER
COMMAND INTERRUPTED
If this message appears repeatedly, please notify the Help Desk.
Enter "HELP STN" for information on contacting the nearest STN Help
Desk by telephone or via SEND in the STNMAIL file.

=> s l1 and (clinical or animal or in adj vivo or mammal or mouse or mice or rat or
monkey or human or rabbit or animal)
L2          43 L1 AND (CLINICAL OR ANIMAL OR IN ADJ VIVO OR MAMMAL OR MOUSE OR
               MICE OR RAT OR MONKEY OR HUMAN OR RABBIT OR ANIMAL)

=> dup rem
ENTER L# LIST OR (END):l2
PROCESSING COMPLETED FOR L2
L3           28 DUP REM L2 (15 DUPLICATES REMOVED)

=> d title l3 1-28
'TITLE' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):scan
'SCAN' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):free
'FREE' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):trial
NO VALID FORMATS ENTERED FOR FILE 'BIOSIS'
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In a multifile environment, each file must have at least one valid format requested. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):bib

L3 ANSWER 1 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1
AN 2003:373554 BIOSIS
DN PREV200300373554
TI Acetylcholinesterase promotes neurite elongation, synapse formation, and surface expression of AMPA receptors in hippocampal neurones.
AU Olivera, Silvia; Rodriguez-Ithurralde, Daniel; Henley, Jeremy M. [Reprint Author]
CS MRC Centre for Synaptic Plasticity, Anatomy Department, School of Medical Sciences, University of Bristol, University Walk, Bristol, BS8 1TD, UK
j.m.henley@bris.ac.uk
SO Molecular and Cellular Neuroscience, (May 2003) Vol. 23, No. 1, pp. 96-106. print.
ISSN: 1044-7431 (ISSN print).
DT Article
LA English
ED Entered STN: 13 Aug 2003
Last Updated on STN: 13 Aug 2003

L3 ANSWER 2 OF 28 MEDLINE on STN DUPLICATE 2
AN 2003252566 MEDLINE
DN PubMed ID: 12694947
TI AMPA receptors on developing medial septum/diagonal band neurons are sensitive to early postnatal binge-like ethanol exposure.
AU Hsiao Shu-Huei; Frye Gerald D
CS Department of Medical Pharmacology and Toxicology, Texas A&M University System Health Science Center, College of Medicine MS 1114, College Station, TX 77843-1114, USA.
NC AA 12386 (NIAAA)
SO Brain research. Developmental brain research, (2003 Apr 14) 142 (1) 89-99.
Journal code: 8908639. ISSN: 0165-3806.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200307
ED Entered STN: 20030603
Last Updated on STN: 20030703
Entered Medline: 20030702

L3 ANSWER 3 OF 28 MEDLINE on STN DUPLICATE 3
AN 2003015991 MEDLINE
DN PubMed ID: 12522159
TI Chronic NMDA receptor blockade from birth delays the maturation of NMDA currents, but does not affect AMPA/kainate currents.
AU Colonnese Matthew T; Shi Jian; Constantine-Paton Martha
CS Department of Biology, Department of Brain and Cognitive Science, and McGovern Institute for Brain Research, Massachusetts Institute of Technology, Cambridge 02139, USA.
NC EY-06039 (NEI)
EY-104074 (NEI)
NS-32290 (NINDS)
SO Journal of neurophysiology, (2003 Jan) 89 (1) 57-68.
Journal code: 0375404. ISSN: 0022-3077.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 200303
ED Entered STN: 20030111
Last Updated on STN: 20030327
Entered Medline: 20030326

L3 ANSWER 4 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:205614 BIOSIS
DN PREV200400206130
TI Regulation of receptor trafficking and influence on synaptic plasticity by TNFalpha.
AU Stellwagen, D. [Reprint Author]; Beattie, E. C.; Malenka, R. C. [Reprint Author]
CS Dept. of Psychiatry and Behavioral Sci., Nancy Pritzker Lab., Stanford Med. Sch., Palo Alto, CA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 903.12. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

L3 ANSWER 5 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:201024 BIOSIS
DN PREV200400201582
TI HSV - 1 amplicon - mediated NMDA - NR2D subunit replacement in neonatal **rat** prevents loss of NMDA receptor function and neurotrophin - 3 (NT - 3) signaling in motor neurons.
AU Arvanian, V. L. [Reprint Author]; Bowers, W. J.; Federoff, H. J.; Mendell, L. M. [Reprint Author]
CS Dept. Neurobiol and Behav, SUNY &&ATT Stony Brook,LSB, Stony Brook, NY, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 547.9. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

L3 ANSWER 6 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:197452 BIOSIS
DN PREV200400198011
TI Synaptic plasticity at glutamatergic synapses on dopamine neurons in the ventral tegmental area (VTA) is detected within two hours of amphetamine injection.
AU Faleiro, L. J. [Reprint Author]; Kauer, J. A. [Reprint Author]
CS Molec. Physiol. Pharmacol. Biotech., Brown Univ., Providence, RI, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 319.8. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

L3 ANSWER 7 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:196256 BIOSIS

DN PREV200400196815
TI NMDAR activation during estrogen **treatment** is required to increase NMDAR transmission and LTP at CA3 - CA1 synapses in **rat** hippocampus.
AU Cofer, C. D. [Reprint Author]; Daigre, J. L. [Reprint Author]; McMahon, L. L. [Reprint Author]
CS Dept. Physiol and Biophysics, Univ. Alabama, Birmingham, AL, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 255.3. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

L3 ANSWER 8 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 4
AN 2002:341853 BIOSIS
DN PREV200200341853
TI Activity-dependent change in AMPA receptor properties in cerebellar stellate cells.
AU Liu, Siqiong June; Cull-Candy, Stuart G. [Reprint author]
CS Department of Pharmacology, University College London, Gower Street, London, WC1E 6BT, UK
s.cull-candy@ucl.ac.uk
SO Journal of Neuroscience, (May 15, 2002) Vol. 22, No. 10, pp. 3881-3889. print.
CODEN: JNRSDS. ISSN: 0270-6474.
DT Article
LA English
ED Entered STN: 19 Jun 2002
Last Updated on STN: 19 Jun 2002

L3 ANSWER 9 OF 28 MEDLINE on STN DUPLICATE 5
AN 2003021149 MEDLINE
DN PubMed ID: 12527472
TI Chronic antidepressant **treatment** increases the membrane expression of AMPA receptors in **rat** hippocampus.
AU Martinez-Turrillas Rebeca; Frechilla Diana; Del Rio Joaquin
CS Universidad de Navarra, Facultad de Medicina Dept. de Farmacologia, c/Irunlarrea 1, Pamplona 31008, Spain.
SO Neuropharmacology, (2002 Dec) 43 (8) 1230-7.
Journal code: 0236217. ISSN: 0028-3908.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20030116
Last Updated on STN: 20030331
Entered Medline: 20030328

L3 ANSWER 10 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 6
AN 2002:540929 BIOSIS
DN PREV200200540929
TI Selective enhancement of AMPA receptor-mediated function in hippocampal CA1 neurons from chronic benzodiazepine-**treated** rats.
AU Van Sickle, Bradley J.; Tietz, Elizabeth I. [Reprint author]
CS Department of Pharmacology, Medical College of Ohio, Toledo, OH, 43614, USA
etietz@mco.edu

SO Neuropharmacology, (July, 2002) Vol. 43, No. 1, pp. 11-27. print.
CODEN: NEPHBW. ISSN: 0028-3908.
DT Article
LA English
ED Entered STN: 16 Oct 2002
Last Updated on STN: 16 Oct 2002

L3 ANSWER 11 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:380260 BIOSIS
DN PREV200300380260
TI ACTIVITY - DEPENDENT TRAFFICKING OF **AMPAR** mRNA IN DENDRITES OF CULTURED HIPPOCAMPAL NEURONS.
AU Grooms, S. Y. [Reprint Author]; Carroll, R. C. [Reprint Author]; Zukin, R. S. [Reprint Author]; Bassell, G. J. [Reprint Author]
CS Dept Neurosci, Albert Einstein Col Med, Bronx, NY, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 839.4. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 20 Aug 2003
Last Updated on STN: 20 Aug 2003

L3 ANSWER 12 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:326303 BIOSIS
DN PREV200300326303
TI PERINATAL HYPOXIC SEIZURES INDUCE AMPA RECEPTOR - MEDIATED DOWN REGULATION OF GABAA RECEPTORS VIA CALCINEURIN ACTIVATION.
AU Dai, W. [Reprint Author]; Lippman, J. J. [Reprint Author]; Jensen, F. E. [Reprint Author]
CS Dept Neurol, Children's Hosp and Harvard Med Sch., Boston, MA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 742.6. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

L3 ANSWER 13 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:325101 BIOSIS
DN PREV200300325101
TI MOLECULAR MECHANISM FOR RAPID OCULAR DOMINANCE PLASTICITY IN VISUAL CORTEX.
AU Yoon, B. J. [Reprint Author]; Heynen, A. J. [Reprint Author]; Liu, C. H. [Reprint Author]; Chung, H.; Huganir, R. L.; Bear, M. F. [Reprint Author]
CS HHMI/Dept Neurosci, Brown Univ, Providence, RI, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 647.12. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

L3 ANSWER 14 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 2003:293913 BIOSIS
DN PREV200300293913
TI CHRONIC NMDA **TREATMENT** AND SUSCEPTIBILITY TO NMDA MEDIATED POTENTIATION IN THE DEVELOPING SUPERIOR COLLICULUS.
AU Zhao, J. [Reprint Author]; Constantine-Paton, M. [Reprint Author]
CS Biology, Brain and Cognitive Sciences, McGovern Institute for Brain Research, MIT, Cambridge, MA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 331.6. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003

L3 ANSWER 15 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:22913 BIOSIS
DN PREV200200022913
TI Calpain and caspase-3 inhibitors prevent L-glutamic acid induced apoptosis and preserve normal electrophysiology in **rat** cortical neurons.
AU Boggan, W. O. [Reprint author]; Ray, S. K.; Nowak, M. W. [Reprint author]; Banik, N. L.
CS Ctr Drug and Alcohol Prg, Med Univ South Carolina, Charleston, SC, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2584. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 26 Dec 2001
Last Updated on STN: 25 Feb 2002

L3 ANSWER 16 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:4026 BIOSIS
DN PREV200200004026
TI Presynaptic terminals undergo functional maturation following brief neurotrophin exposure.
AU Renger, J. J. [Reprint author]; Rao, V. [Reprint author]; Li, B. [Reprint author]; Liu, G. [Reprint author]
CS Brain and Cognitive Sciences, Mass Inst Tech, Cambridge, MA, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2396. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Dec 2001
Last Updated on STN: 25 Feb 2002

L3 ANSWER 17 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:574525 BIOSIS
DN PREV200100574525
TI Upregulation of GABAA, but not AMPA, kainate or NMDA receptor expression in single CA1 pyramidal cells of chronically epileptic rats: DNA array study.
AU Rikhter, T. Y. [Reprint author]; Hsu, F. H. [Reprint author]; Brooks-Kayal, A. R. [Reprint author]; Lynch, D. R. [Reprint author];

Coulter, D. A. [Reprint author]
CS Neurology, Stokes Res Institute, Children's Hosp Philadelphia, Philadelphia, PA, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2079. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Dec 2001
Last Updated on STN: 25 Feb 2002

L3 ANSWER 18 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:509123 BIOSIS
DN PREV200100509123
TI Age-related change in ratio of **AMPAR**- to NMDAR-mediated synaptic transmission in FD is not modified by experience.
AU Yang, Z. [Reprint author]; Krause, M. [Reprint author]; Rao, G.; Houston, F. P. [Reprint author]; White, R. [Reprint author]; McNaughton, B. L. [Reprint author]; Barnes, C. A. [Reprint author]
CS Neural Systems, Memory and Aging, Univ. Arizona, Tucson, AZ, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 834. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002

L3 ANSWER 19 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 7
AN 2001:563142 BIOSIS
DN PREV200100563142
TI Transient synaptic activation of NMDA receptors leads to the insertion of native AMPA receptors at hippocampal neuronal plasma membranes.
AU Pickard, Lisa; Noel, Jacques; Duckworth, Joshua K.; Fitzjohn, Stephen M.; Henley, Jeremy M.; Collingridge, Graham L. [Reprint author]; Molnar, Elek
CS MRC Centre for Synaptic Plasticity, Department of Anatomy, School of Medical Sciences, University of Bristol, University Walk, Bristol, BS8 1TD, UK
g.l.collingridge@bris.ac.uk
SO Neuropharmacology, (November, 2001) Vol. 41, No. 6, pp. 700-713. print.
CODEN: NEPHBW. ISSN: 0028-3908.
DT Article
LA English
ED Entered STN: 5 Dec 2001
Last Updated on STN: 25 Feb 2002

L3 ANSWER 20 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:497731 BIOSIS
DN PREV200100497731
TI NMDA receptor-mediated currents in acutely dissociated hippocampal CA1 neurons after 1 week benzodiazepine **treatment**.
AU Tietz, E. I. [Reprint author]; Van Sickle, B. J. [Reprint author]; Greenfield, L. J. [Reprint author]
CS Pharmacology, Med Col Ohio, Toledo, OH, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 682. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 24 Oct 2001
Last Updated on STN: 23 Feb 2002

L3 ANSWER 21 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:478235 BIOSIS
DN PREV200100478235
TI Carboxyfullerene C3 prevents AMPA excitotoxicity in dopaminergic neurons in culture.
AU de Erausquin, G. A. [Reprint author]; Hyrc, K. L. [Reprint author]; Yamada, K. A. [Reprint author]; Dugan, L. L. [Reprint author]; Goldberg, M. P. [Reprint author]
CS Neurology Dept, CSNSI, Washington University, Saint Louis, MO, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 271. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 10 Oct 2001
Last Updated on STN: 23 Feb 2002

L3 ANSWER 22 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 8
AN 2001:535999 BIOSIS
DN PREV200100535999
TI Visual-mediated regulation of retinal CaMKII and its GluR1 substrate is age-dependent.
AU Xue, Jin; Li, Guangyu; Laabich, Aicha; Cooper, Nigel G. F. [Reprint author]
CS Department of Ophthalmology and Visual Sciences, University of Louisville School of Medicine, 500 South Preston Street, Louisville, KY, 40202, USA
nigelcooper@louisville.edu
SO Molecular Brain Research, (10 September, 2001) Vol. 93, No. 1, pp. 95-104. print.
CODEN: MBREE4. ISSN: 0169-328X.
DT Article
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002

L3 ANSWER 23 OF 28 MEDLINE on STN DUPLICATE 9
AN 2001189976 MEDLINE
DN PubMed ID: 11277968
TI Interferon-gamma-induced changes in synaptic activity and AMPA receptor clustering in hippocampal cultures.
AU Vikman K S; Owe-Larsson B; Brask J; Kristensson K S; Hill R H
CS Department of Neuroscience, Nobels Vag 12A, Karolinska Institutet, SE-171 77, Stockholm, Sweden.. kristina.vikman@neuro.ki.se
SO Brain research, (2001 Mar 30) 896 (1-2) 18-29.
Journal code: 0045503. ISSN: 0006-8993.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200106
ED Entered STN: 20010702
Last Updated on STN: 20010702
Entered Medline: 20010628

L3 ANSWER 24 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 2001:108127 BIOSIS
DN PREV200100108127
TI Activity co-regulates AMPA and NMDA synaptic currents in cortical pyramidal neurons.
AU Watt, A. J. [Reprint author]; Nelson, S. B.; Turrigiano, G. G.
CS Brandeis University, Waltham, MA, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-521.7. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Feb 2001
Last Updated on STN: 15 Feb 2002

L3 ANSWER 25 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:108932 BIOSIS
DN PREV200100108932
TI Altered GABAA, AMPA, KA and NMDA receptor subunit mRNA expression in single dentate granule cells of pilocarpine-**treated** rats before the onset of epilepsy.
AU Rikhter, T. Y. [Reprint author]; Shumate, M. D.; Brooks-Kayal, A. R.; Jin, H.; Lynch, D. R.; Coulter, D. A.
CS Children's Hosp Philadelphia, University of Pennsylvania, Philadelphia, PA, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-620.7. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Feb 2001
Last Updated on STN: 15 Feb 2002

L3 ANSWER 26 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:87884 BIOSIS
DN PREV200100087884
TI Behavioral sensitization to cocaine is associated with changes in nucleus accumbens excitatory synaptic transmission.
AU Thomas, M. J. [Reprint author]; Malenka, R. C.
CS Stanford University, Palo Alto, CA, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-292.3. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Feb 2001
Last Updated on STN: 12 Feb 2002

L3 ANSWER 27 OF 28 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:97318 BIOSIS
DN PREV200100097318
TI Changes in AMPA receptor antagonist binding and subunit protein immunostaining in benzodiazepine-tolerant **rat**.
AU VanSickle, B. J. [Reprint author]; Lilly, S. M.; Tietz, E. I.
CS Medical College of Ohio, Toledo, OH, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract

No.-425.6. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 21 Feb 2001
Last Updated on STN: 15 Feb 2002

L3 ANSWER 28 OF 28 MEDLINE on STN DUPLICATE 10
AN 2000029305 MEDLINE
DN PubMed ID: 10565575
TI Epileptiform propagation patterns mediated by NMDA and non-NMDA receptors in **rat** neocortex.
AU Telfeian A E; Connors B W
CS Department of Neuroscience, Brown University, Providence, Rhode Island 02912, USA.
SO Epilepsia, (1999 Nov) 40 (11) 1499-506.
Journal code: 2983306R. ISSN: 0013-9580.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199911
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991126

=> d his

(FILE 'HOME' ENTERED AT 08:47:04 ON 10 JUN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:47:18 ON 10 JUN 2004
L1 57 S (AMPA ADJ RECEPTOR OR AMPAR) AND TREAT?
L2 43 S L1 AND (CLINICAL OR ANIMAL OR IN ADJ VIVO OR MAMMAL OR MOUSE
L3 28 DUP REM L2 (15 DUPLICATES REMOVED)

=> l1 not l3
L1 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l1 not l3
L4 29 L1 NOT L3

=> dup rem
ENTER L# LIST OR (END):l4
PROCESSING COMPLETED FOR L4
L5 19 DUP REM L4 (10 DUPLICATES REMOVED)

=> d ibib l5 1-5

L5 ANSWER 1 OF 19 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003272790 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12799140
TITLE: Acetylcholinesterase promotes neurite elongation, synapse formation, and surface expression of AMPA receptors in hippocampal neurones.
AUTHOR: Olivera Silvia; Rodriguez-Ithurralde Daniel; Henley Jeremy M
CORPORATE SOURCE: MRC Centre for Synaptic Plasticity, Anatomy Department,

School of Medical Sciences, University of Bristol, University Walk, UK.
SOURCE: Molecular and cellular neurosciences, (2003 May) 23 (1) 96-106.
Journal code: 9100095. ISSN: 1044-7431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030612
Last Updated on STN: 20030729
Entered Medline: 20030728

L5 ANSWER 2 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
ACCESSION NUMBER: 2003:256391 BIOSIS
DOCUMENT NUMBER: PREV200300256391
TITLE: AMPA receptors on developing medial septum/diagonal band neurons are sensitive to early postnatal binge-like ethanol exposure.
AUTHOR(S): Hsiao, Shu-Huei; Frye, Gerald D. [Reprint Author]
CORPORATE SOURCE: Department of Medical Pharmacology and Toxicology, College of Medicine, Texas A and M University System Health Science Center, MS 1114, College Station, TX, 77843-1114, USA
gdfrye@tamu.edu
SOURCE: Developmental Brain Research, (14 April 2003) Vol. 142, No. 1, pp. 89-99. print.
CODEN: DBRRDB. ISSN: 0165-3806.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 May 2003
Last Updated on STN: 28 May 2003

L5 ANSWER 3 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3
ACCESSION NUMBER: 2003:99025 BIOSIS
DOCUMENT NUMBER: PREV200300099025
TITLE: Chronic NMDA receptor blockade from birth delays the maturation of NMDA currents, but does not affect AMPA/kainate currents.
AUTHOR(S): Colonnese, Matthew T.; Shi, Jian; Constantine-Paton, Martha [Reprint Author]
CORPORATE SOURCE: Massachusetts Institute of Technology, 77 Massachusetts Avenue, Building 68-380, Cambridge, MA, 02139-4307, USA
mcpaton@mit.edu
SOURCE: Journal of Neurophysiology (Bethesda), (January 2003) Vol. 89, No. 1, pp. 57-68. print.
ISSN: 0022-3077 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Feb 2003
Last Updated on STN: 12 Feb 2003

L5 ANSWER 4 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:205483 BIOSIS
DOCUMENT NUMBER: PREV200400205999
TITLE: Localized upregulation of AMPA GluR1 subunit in hiipocampal CA1 neurons after 1 - week benzodiazepine **treatment**
.
AUTHOR(S): Tietz, E. I. [Reprint Author]; Lilly, S. M. [Reprint Author]; Alvarez, F. J.; Grounds, K. M. [Reprint Author]; Song, J. [Reprint Author]
CORPORATE SOURCE: Dept. Pharmacol, Med. Col. Ohio, Toledo, OH, USA

SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 895.1. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

L5 ANSWER 5 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:205486 BIOSIS
DOCUMENT NUMBER: PREV200400206002
TITLE: Possible increased expression of calcium permeable AMPA/kainate (Ca - A/K) channels in hippocampal pyramidal neurons by tumor necrosis factor - alpha (TNF - alpha) .
AUTHOR(S): Ogoshi, F. [Reprint Author]; Yin, H. Z.; Song, B.; Weiss, J. H.
CORPORATE SOURCE: Anat. and NeuroBiol., UC Irvine, Irvine, CA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 895.4. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

=> d his

(FILE 'HOME' ENTERED AT 08:47:04 ON 10 JUN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:47:18 ON 10 JUN 2004
L1 57 S (AMPA ADJ RECEPTOR OR AMPAR) AND TREAT?
L2 43 S L1 AND (CLINICAL OR ANIMAL OR IN ADJ VIVO OR MAMMAL OR MOUSE
L3 28 DUP REM L2 (15 DUPLICATES REMOVED)
L4 29 S L1 NOT L3
L5 19 DUP REM L4 (10 DUPLICATES REMOVED)

=> s (AMPA adj receptor or AMPAR) AND inhibit?
L6 164 (AMPA ADJ RECEPTOR OR AMPAR) AND INHIBIT?

=> s l6 and treat?
L7 17 L6 AND TREAT?

=> dup rem
ENTER L# LIST OR (END):l7
PROCESSING COMPLETED FOR L7
L8 13 DUP REM L7 (4 DUPLICATES REMOVED)

=> d l8 1-13 abs

L8 ANSWER 1 OF 13 MEDLINE on STN DUPLICATE 1
AB The impact of binge-like, early postnatal ethanol **treatment** on AMPA or kainate whole cell currents was examined in acutely isolated medial septum/diagonal band (MS/DB) neurons. AMPA (10 or 100 microM) current was **inhibited** by GYKI 52466, a selective AMPA receptor (**AMPAR**) antagonist, in all neurons isolated on postnatal day (PD)

5-8, PD 12-15 or PD 32-35. Cyclothiazide, a selective **inhibitor** of **AMPAR** desensitization, also effectively potentiated AMPA currents. This suggests that non-NMDA, ionotropic glutamate receptors on immature MS/DB neuron are predominantly AMPARs. Concentration-dependent kainate (10-1000 microM) application evoked nondesensitizing currents that exhibited an increase in the maximum response by the end of first postnatal month, consistent with developmental regulation of **AMPAR** function. Acute 3 s ethanol application (100 mM) consistently blunted AMPA- and kainate currents approximately 20-30% across age groups. **Inhibition** was sustained during continuous ethanol superfusion lasting 10-12 min without evidence of acute tolerance. Repeated oral intubation of rat pups with ethanol (5.25 g/kg/day on PD 4-9), which models third trimester human binge drinking, resulted in peak blood ethanol levels of approximately 350 mg/dl (measured 90 min after PD 6 dosing). AMPA or kainate currents were upregulated in neurons isolated on PD 32-35 by earlier ethanol intubation suggesting that binge-like intoxication augments developing **AMPAR** function. Despite this augmentation of **AMPAR** function, no significant changes were found in the sensitivity of AMPA currents to GYKI 52466, cyclothiazide or acute ethanol (100 mM) sensitivity or in the levels of GluR1/GluR2 subunit proteins from MS/DB tissue. These results indicate that non-NMDA ionotrophic glutamate receptors on immature MS/DB neurons, which are largely of the **AMPAR** subtype, are moderately sensitive to immediate **inhibition** by ethanol. Repeating this **inhibition** during early postnatal binge-like intoxication can augment normal development of **AMPAR** function.

L8 ANSWER 2 OF 13 MEDLINE on STN

AB The activity of the N-methyl-D-aspartate receptor (NR) regulates the composition of excitatory synapses and mediates multiple forms of synaptic and structural plasticity. In the superficial superior colliculus (sSC) of the rat, NR activity is essential for the full refinement of retinotopy during development. We have examined the NR's role in synaptic development by chronically **treating** the sSC from birth with the competitive antagonist (+/-)-2-amino-5-phosphonopentanoic acid (AP5) released by the slow-release polymer Elvax. Whole-cell voltage-clamp recordings were used to characterize excitatory postsynaptic potentials (EPSCs) in slices from postnatal day (P)12-20 sSC. Chronic NR blockade reduced the ratio of AMPA/kainate receptor (**AMPAR**) to NR peak current amplitudes of both spontaneous (s)EPSCs and evoked EPSCs. Spontaneous NR current amplitude was increased following **treatment**, while spontaneous **AMPAR** currents were identical to those of controls, indicating that the ratio change was due to an increased NR current. Comparison of sEPSC frequency, **AMPAR** current rectification, and quantitative Western blots indicated that the characteristics of AMPARs at the synapse are normal following AP5 **treatment**. In the sSC, NR currents show a rapid decrease in decay time on P11 and previous studies in slices indicate this change results from a NR-mediated activation of the phosphatase calcineurin. Consistent with this in vitro finding, the down-regulation failed to occur in sSC chronically **treated** with AP5 in vivo. Together the present data show that NR function is necessary for subsequent NR current regulation in vivo, but it is not essential for the developmental expression of normal **AMPAR** currents.

L8 ANSWER 3 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AB Regulation of the AMPA subtype of glutamate receptor is believed to contribute to long-term changes in synaptic strength; changes thought to be important for many forms of experience-dependent plasticity. Tumor Necrosis Factor-alpha (TNF) has been shown to induce a rapid exocytosis of AMPA receptors (AMPARs) in cultured hippocampal neurons, and its constitutive release in both cultures and intact hippocampal slices appears to be required for maintaining AMPARs on the cell surface (Beattie, et al., 2002, Science, 295:2282). Here we present a further

examination of the neuronal effects of TNF on cultured hippocampal neurons. To assay the specificity of TNF action on receptor trafficking, we examined its effects on the surface expression of several receptors, including the GABA-A receptor. Application of TNF did not result in the exocytosis of GABA-A receptors, and initial experiments suggest that in fact TNF caused a decrease in the surface expression of GABA-A receptors and enhanced their constitutive endocytosis. We also have investigated the identity of the TNF-activated intracellular signaling cascades that result in the exocytosis of AMPARs. Examination of the effects of **inhibitors** of various intracellular signaling pathways (i.e. PKA, CaMKII, p38 and P42/44MAPK, PI-3 kinase) suggests that PI-3 kinase is required for the effects of TNF on **AMPAR** trafficking while other pathways are dispensable. Lastly, we examined the interaction of TNF with synaptic plasticity in hippocampal slices. Initial experiments suggest that LTP was impaired in slices prepared from TNF knockout mice while LTD appeared to be unaffected. We also examined the consequences of acutely **treating** slices with TNF and these results will be presented.

L8 ANSWER 4 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB The action of glutamate is mediated by the activation of metabotropic (mGluRs) and ionotropic (iGluRs) receptors in the CNS. The mGluRs are highly enriched in prefrontal cortex (PFC)-a brain region critically involved in the regulation of cognition and emotion. Emerging evidence has suggested that mGluRs are viable drug targets for neuropsychiatric disorders associated with PFC dysfunction. However the mGluR-mediated signaling in PFC remains unclear. To understand the physiological functions of group II mGluR (mGluR 2/3) in PFC neurons, we investigated the cellular and molecular mechanisms underlying the actions of group II mGluRs on ligand-gated ion channels. We found that APDC, a highly selective and potent group II mGluR agonist, reversibly enhanced NMDAR currents in acutely dissociated PFC pyramidal neurons, while it had no effect on GABAAR or **AMPAR** currents. The mGluR2/3 antagonists APICA and LY 341493 blocked APDC-induced enhancement of NMDAR currents, suggesting the mediation by mGluR 2/3 receptors. This APDC effect on NMDARs was largely blocked by dialysis with the Ca2+ chelator or the **inhibition** of protein kinase C (PKC). In contrast, **inhibiting** CaMKII, or protein tyrosine kinases, or cycline-dependent kinase 5 (Cdk 5) failed to alter the APDC effect. Moreover, APDC increased the PKC activity in PFC slices. These findings suggest that activation of mGluR2/3 receptors potentiates NMDAR channel functions in PFC through a mechanism involving Ca2+ and PKC. This modulation may be relevant for developing novel mGluR-related pharmacological agents for the **treatment** of mental illnesses.

L8 ANSWER 5 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Ovariectomized (OVX) rats **treated** with 17beta estradiol (E2) have increased dendritic spine density (Woolley 1999) and LTP magnitude (Cordoba Montoya 1997) at CA3-CA1 synapses. E2 also decreases GABAergic **inhibition** and increases NMDAR transmission (Rudick 2001). We have previously shown that E2 increases LTP at 24,48 but not 72 hours following estrogen **treatment**. Additionally, we have shown that NMDAR activation is required during E2 **treatment** to increase LTP, similarly to the E2-induced increase in spine density. The goal of this study was to test whether the E2-induced increase in NMDAR transmission requires NMDAR activation during E2 **treatment** and whether the magnitude of LTP at 72 hours is due to an increase in **inhibition**. We used standard CA1 dendritic field potential recordings in acute hippocampal slices from 7-9 week OVX rats **treated** with either E2(10mg/d, twice, 24 hours apart) or oil vehicle 10-12 days after OVX. Stimulus response curves performed in slices from E24 and control animals show that E2 selectively increases NMDAR transmission, which is correlated with the increase in LTP ($p<0.05$). Blockade of NMDARs during E2 **treatment** with MK-801 (0.2mg/kg/d) blocks the increase in NMDAR transmission, the increase in LTP and the

increase in spine density (p>0.05). Additionally, acute blockade of GABAARs with picrotoxin does not increase LTP at 72 hours, indicating that the decreased LTP at this time point is not due to an increase in **inhibition** (72 141+5%; C 128+5% p>0.05). However, at 72 hours, both NMDAR and **AMPAR** transmission are increased (p<0.05). This data suggests the increase in LTP at 24 hours may be due to newly formed NMDAR-only synapses which are converted to active synapses by 72 hours, as indicated by the increase in **AMPAR** in addition to NMDAR transmission at this time point.

L8 ANSWER 6 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Stargazin (stg) is a transmembrane protein that increases AMPA receptor (**AMPAR**) surface expression. To clarify the mechanism for this effect, we measured the ratio of surface to internal (S/I) GluR1 in transfected COS7 cells using biochemical and immunocytochemical assays. Stg transfection strongly increased GluR1 S/I. This effect was not reduced by cotransfection of a dominant-negative dynamin mutant, indicating that stg's mechanism of action did not involve **inhibition** of **AMPAR** endocytosis. To explore for a possible chaperone function of stg, we **treated** cells with drugs known to upregulate endoplasmic reticulum (ER) chaperones as part of the unfolded protein response (UPR). The proteasome **inhibitors** MG132 and lactacystin, which induce the UPR by blocking ER-associated degradation of misfolded proteins, caused a dose-dependent increase in GluR1 S/I without increasing total GluR1 levels. This effect was nearly maximal within 2 h of exposure, and was prevented by cotreatment with cycloheximide. Importantly, this effect partially mimicked and occluded the effect of stg, suggesting a common mechanism of action. In contrast, agents that induce the UPR by interfering with proper protein folding, such as tunicamycin, thapsigargin and dithiothreitol, decreased GluR1 S/I. These data support a model where stg increases surface levels of AMPA receptors by acting as a specific chaperone.

L8 ANSWER 7 OF 13 MEDLINE on STN DUPLICATE 2
AB Two days following one-week administration of the benzodiazepine, flurazepam (FZP), rats exhibit anticonvulsant tolerance in vivo, while reduced GABA(A) receptor-mediated **inhibition** and enhanced EPSP amplitude are present in CA1 pyramidal neurons in vitro. AMPA receptor (**AMPAR**)-mediated synaptic transmission in FZP-**treated** rats was examined using electrophysiological techniques in in vitro hippocampal slices. In CA1 pyramidal neurons from FZP-**treated** rats, the miniature excitatory postsynaptic current (mEPSC) amplitude was significantly increased (33%) without change in frequency, rise time or decay time. Moreover, mEPSC amplitude was not elevated in dentate granule neurons following 1-week FZP **treatment** or in CA1 pyramidal neurons following acute desalkyl-FZP **treatment**. Regulation of **AMPAR** number was assessed by quantitative autoradiography with the **AMPAR** antagonist, [(3)H]Ro48-8587. Specific binding was significantly increased in stratum pyramidale of hippocampal areas CA1 and CA2 and in proximal dendritic fields of CA1 pyramidal neurons. Regulation of **AMPAR** subunit proteins was examined using immunological techniques. Neither abundance nor distribution of GluR1-3 subunit proteins was different in the CA1 region following FZP **treatment** . These findings suggest that enhanced **AMPAR** currents, mediated at least in part by increased **AMPAR** number, may contribute to BZ anticonvulsant tolerance. Furthermore, these studies suggest an interaction between GABAergic and glutamatergic systems in the CA1 region which may provide novel therapeutic strategies for restoring BZ effectiveness.

L8 ANSWER 8 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Hypoxia is the most common cause of perinatal seizures and can be associated with long term hyperexcitability. In a rodent model, hypoxica causes seizures at postnatal day (P) 10-12, and is associated with long

term increases in seizure susceptibility. We reported that Ca2+-permeable AMPA receptor (**AMPAR**) activation is required to initiate hypoxic seizures, and also an early decrease in GABAA receptor (GABAAR) IPSCs (Sanchez, et al., Soc Neurosci. Absolute 2001). GABARs can be downregulated by dephosphorylation by Ca2+/CaM dependent phosphatase calcineurin (CaN), and CaN expression is increased following perinatal hypoxia. We investigated the contribution of AMPARs and CaN in the decreased GABAR function in this model. Hippocampal slices were prepared from P 10-11 rats after hypoxia-induced seizures (4-7% of O2,15 min). Whole-cell patch-clamp recordings were made in CA1 pyramidal neurons in slices from hypoxia-**treated** and age-matched control rats. Both the frequency (0.820.1 Hz) and amplitude (12.91.4 pA) of spontaneous GABAAR ISPCs (sIPSCs) after hypoxia induced seizures were significantly decreased compared to controls (1.70.2 Hz, $p<0.01$ and 19.11.5 pA, $p<0.05$). FK-506 reversed the hypoxia-induced attenuation of sIPSCs frequency (1.80.2 Hz, $p<0.01$). Similarly, sIPSC frequency was markedly increased with blockade of AMPARs and NMDARs by CNQX and APV. CNQX alone increased sIPSC frequency (3.40.2 Hz, $p<0.001$), while APV showed a nonsignificant increase. These data suggest that Ca2+-permeable AMPARs may activate CaN and contribute to the early decreases in GABAAR **inhibition** and contribute to the epileptogenic effects of perinatal hypoxia.

L8 ANSWER 9 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AB Two days after 1-week **treatment** with the benzodiazepine (BZ), flurazepam (FZP), rats display anticonvulsant tolerance in vivo. Concurrently, CA1 pyramidal neurons in hippocampal slices show reduced GABAA receptor (GABAR)-mediated **inhibition**. Interestingly, AMPA receptor (**AMPAR**)-mediated mEPSC amplitude is increased while evoked NMDA receptor (NMDAR) EPSC amplitude is reduced. Whole-cell slice recordings in CA1 neurons examined the temporal relationship between excitation/**inhibition** at 0, 1 and 4 days after FZP **treatment**. At 0 days, neither **AMPAR** mEPSC nor evoked NMDAR EPSC amplitudes were altered. At 1 day, **AMPAR** mEPSC amplitude was significantly increased (CON: -10.9+-0.4 pA; FZP: -12.3+-0.3 pA; +12%; $p<0.01$) but was less than previously found at 2 days (+33%). No change in NMDAR EPSC or GABAR mIPSC amplitude was found, unlike at 2 days. However, in vitro tolerance to zolpidem (1 muM) prolongation of mIPSC decay (CON: 139%; FZP: 112%; $p<0.01$) was present suggesting BZ tolerance in the absence of reduced GABAR mIPSC amplitude. No changes in excitation were found at 4 days. A possible consequence of altered excitation, in vivo, was examined by elevated plus maze 0, 1, and 2 days after FZP **treatment**. Increased anxiety at 1 day suggests a role for increased **AMPAR** function in BZ dependence, while lack of anxiety at 2 days suggests decreased NMDAR function (50%) may compensate for increased **AMPAR** function and prevent anxiety. BZ tolerance and dependence may reflect temporally altered excitation/**inhibition** within specific brain regions. Whether each phenomenon arises from linked or separable mechanisms is currently under investigation.

L8 ANSWER 10 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AB The release of L-glutamic acid (LGA) in CNS injury and diseases causes neuronal death and dysfunction. In an in vitro model using rat primary cortical neurons, we tested the efficacy of calpain and caspase-3 **inhibitors** alone and in combination to prevent neuronal death and preserve physiological function following exposure to LGA. Cortical neurons exposed to 0.5 muM LGA for 24 h committed mostly apoptotic death as detected by Wright staining and ApopTag assay. Also, in situ double labeling identified active caspase-3-p20 fragment and DNA fragmentation in apoptotic neurons. Pre-**treatment** of neurons with 0.2 muM calpeptin (calpain-specific **inhibitor**) or/and 100 muM z-DEVD-fmk (caspase-3-specific **inhibitor**) prevented apoptosis. Electrophysiological properties (resting membrane potential, leak current at -70 mV and whole-cell capacitance) and whole-cell currents associated with voltage-gated Na+ channels, AMPARs and NMDARs were measured. The

lack of a change in capacitance indicated that neurons **treated** with **inhibitor**(s) and LGA did not undergo apoptotic shrinkage and maintained the same size as the control neurons. Currents associated with Na+ channels, AMPARs, and NMDARs were similar in amplitude and activation/inactivation kinetics for control and all **treatments** with **inhibitor**(s) and LGA. Spontaneous synaptic activity as oberved by miniature end-plate currents was also similar. Therefore, prevention of LGA induced apoptosis by protease **inhibitors** resulted in neurons with normal electrophysiological properties and ion channel activity.

L8 ANSWER 11 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Temporal lobe epilepsy is associated with alterations in neurotransmitter receptor function and mRNA expression in various populations of hippocampal neurons. Epilepsy-associated alterations in GABAA receptor (GABAR) function have been identified in CA1 cells in chronically epileptic pilocarpine-**treated** rats (Gibbs et al., 1997). To investigate the expression of major **inhibitory** and excitatory receptors in this model of epilepsy, we examined GABAR, AMPA (**AMPAR**), KA (KAR) and NMDA (NMDAR) receptor subunit mRNA profiles in individual acutely isolated CA1 pyramidal cells from control (n=9 cells, 6 animals) and epileptic animals (n=10/2) using the single cell aRNA amplification method. All cells exhibited viable GABA currents. GABAR (alpha1-6, beta1-3, gamma1-3, delta), **AMPAR** (GluR1-4), KAR (GluR5-7, KA1-2) and NMDAR (NR1, NR2A, NR2B, NR2C, NR2D) subunit mRNAs were profiled. Total GABAR mRNA expression was significantly increased relative to NFL (to 156% of control, P<0.02) in CA1 cells from epileptic rats. Expression levels of **AMPAR**, KAR and NMDAR were not different from control in epileptic CA1 neurons. Therefore, the increase in GABAR mRNA is selective and contrasts with the unchanged total mRNA expression for all other profiled receptors. In CA1 cells from chronically epileptic animals, alterations in GABAR expression and function may contribute disproportionately to alterations in excitability since transcriptional production of most other receptors is unchanged.

L8 ANSWER 12 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB GABA **inhibition** is reduced (50%) in CA1 neurons 2 days after ending chronic benzodiazepine (BZ) **treatment**. AMPA receptor (**AMPAR**) function, i.e. mEPSC amplitude, is concomitantly increased (25%) consistent with a localized increase in **AMPAR** binding. Additionally, downregulation of NR2B subunit mRNA levels were mirrored by a reduction in NR2B protein levels suggesting that NMDAR function may also be altered. CA1 neurons were acutely dissociated from rat hippocampus 2 days after 1 week flurazepam (FZP) administration (100 mg/kgX3 days; 150 mg/kgX4 days, p.o.). Cells were recorded (Axopatch 200B) in a no added Mg2+ external solution and an internal solution containing CsCH3SO3 and an ATP/GTP regeneration system. Cells were voltage clamped at -30 mV to obviate any effects of ambient Mg2+ and to allow GABA responses to be recorded. Drug was applied with a 'multipuffer' U-tube device in increasing concentrations (GABA 10muM; NMDA 0.3 to 1000 muM). GABA induced outward currents in all cells tested. NMDA induced concentration-dependent inward currents in control cells (EC50=83 muM; Imax=362.5+-5.4 pA; n=6). There was a 1.7 fold rightward shift of the concentration-response (C-R) curve in FZP-**treated** cells and a 20% decrease in the maximum current (EC50=142 muM; Imax=290.6+-7.5 pA; n=8). The use-dependent activation of BZ receptors, in addition to reducing GABA **inhibition**, downregulates NMDAR and upregulates **AMPAR** function suggesting a significant excitatory-**inhibitory** imbalance in BZ-**treated** CA1 neurons that may contribute to functional BZ tolerance.

L8 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Protracted activation of AMPA receptors (**AMPAr**) causes selective pruning and cell death of dopaminergic neurons (DNs) in vitro by a

mechanism involving reactive oxygen species (Soc Neursci Abs 1999, 820.1). We tested the effect of the potent free radical scavenger carboxylullerene C3 (C3) (PNAS, 1997; 94:9434) on **AMPAr** mediated toxicity. Primary cultures of rat cortex or mesencephalon were prepared from e-14 embryos, and maintained for 14 days. Cultures **treated** with AMPA or Kainate (100 muM) for 24h, and immunostained with for Tyrosine Hydroxylase and/or MAP-2 for DNs counting and morphometric studies, revealed 50% loss of DNs, and loss of neurites and synaptic contacts (by synaptophysin immunostaining) in surviving neurons. Combined with 100 muM AMPA or Kainate, 30 muM C3 prevented DNs death, neurite pruning, and loss of synaptic contacts. Loss of neurite function as reflected by 3H-dopamine uptake was also prevented by C3 **treatment**. A possible direct effect of C3 on **AMPAr** was assessed by single cell imaging (with sodium-sensitive ratio-metric dye SBFI) and whole-cell patch clamp electrophysiology in cortical cultures. Preincubation with C3 blocked the kainate-induced current and caused a marked reduction in kainate-induced sodium influx. However, coapplication of C3 reduced both currents and sodium influx only to a small degree. These results suggest that C3 maybe a useful neuroprotectant for DNs. Blockade of **AMPAr** may add to the well characterized efects of C3 on membrane properties and ROS, or be an indirect consequence of the latter.

```
=> s l6 and antagonist or quinazolin?
<------User Break------>

SEARCH ENDED BY USER

=> s l6 and (antagonist or quinazolin?)
L9          38 L6 AND (ANTAGONIST OR QUINAZOLIN?)

=> s l9 and (treat? or rat or monkey or animal or (in adj vivo))
L10         30 L9 AND (TREAT? OR RAT OR MONKEY OR ANIMAL OR (IN ADJ VIVO))

=> dup rem
ENTER L# LIST OR (END):l10
PROCESSING COMPLETED FOR L10
L11          21 DUP REM L10 (9 DUPLICATES REMOVED)

=> d l11 bib abs 1-21

L11  ANSWER 1 OF 21  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
AN   2004114243  EMBASE
TI   Two Loci of Expression for Long-Term Depression at Hippocampal Mossy
     Fiber-Interneuron Synapses.
AU   Lei S.; McBain C.J.
CS   C.J. McBain, Lab. of Cell./Synaptic Neurophysiol., National Institutes of
     Health, Building 49, Convent Drive, Bethesda, MD 20892, United States.
     mcbainc@mail.nih.gov
SO   Journal of Neuroscience, (3 Mar 2004) 24/9 (2112-2121).
     Refs: 60
     ISSN: 0270-6474  CODEN: JNRSDS
CY   United States
DT   Journal; Article
FS   002     Physiology
     008     Neurology and Neurosurgery
LA   English
SL   English
AB   Two distinct forms of long-term depression (LTD) exist at mossy fiber
     synapses between dentate gyrus granule cells and hippocampal CA3 stratum
     lucidum interneurons. Although induction of each form of LTD requires an
     elevation of postsynaptic intracellular Ca(2+), at Ca (2+)-impermeable
     AMPA receptor (CI-AMPAR) synapses, induction is NMDA receptor
```

(NMDAR) dependent, whereas LTD at Ca(2+)-permeable AMPA receptor (CP-**AMPAR**) synapses is NMDAR independent. However, the expression locus of either form of LTD is not known. Using a number of criteria, including the coefficient of variation, paired-pulse ratio, AMPA-NMDA receptor activity, and the low-affinity **AMPAR antagonist** γ-D-glutamyl-glycine, we demonstrate that LTD expression at CP-**AMPAR** synapses is presynaptic and results from reduced transmitter release, whereas LTD expression at CI-**AMPAR** synapses is postsynaptic. The N-ethylmaleimide-sensitive fusion protein-AP2-dathrin adaptor protein 2 **inhibitory** peptide pep2m occluded LTD expression at CI-**AMPAR** synapses but not at CP-**AMPAR** synapses, confirming that CI-**AMPAR** LTD involves postsynaptic **AMPAR** trafficking. Thus, mossy fiber innervation of CA3 stratum lucidum interneurons occurs via two parallel systems targeted to either Ca(2+)-permeable or Ca(2+)-impermeable AMPA receptors, each with a distinct expression locus for long-term synaptic plasticity.

L11 ANSWER 2 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

AN 2003304945 EMBASE

TI Desensitization of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors facilitates use-dependent **inhibition** by pentobarbital.

AU Jackson M.F.; Joo D.T.; Al-Mahrouki A.A.; Orser B.A.; Macdonald J.F.

CS Dr. M.F. Jackson, Department of Physiology, Medical Sciences Bldg., University of Toronto, 1 King's College Circle, Toronto, Ont. M5S 1A8, Canada. mike.jackson@utoronto.ca

SO Molecular Pharmacology, (1 Aug 2003) 64/2 (395-406).
Refs: 40
ISSN: 0026-895X CODEN: MOPMA3

CY United States

DT Journal; Article

FS 024 Anesthesiology
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB Although the mechanisms underlying the use-dependent **inhibition** of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (AMPARs) by barbiturates are not well understood, it has generally been assumed to involve open channel block. We examined the properties of the **inhibition** of AMPARs by the barbiturate pentobarbital (PB) in acutely isolated and cultured hippocampal neurons. PB caused a use- and concentration-dependent **inhibition** (IC(50) = 20.7 μM) of **AMPAR**-mediated currents evoked by kainate. Contrary to the properties of an open channel blocker, the **inhibition** by PB developed with double exponential kinetics was reduced under conditions that favor the open channel state of AMPARs and was independent of membrane voltage. In addition, the **inhibition** was reduced at basic pH, indicating that the uncharged form of PB is active at AMPARs. Preventing **AMPAR** desensitization with cyclothiazide reduced the potency of **inhibition** by PB and prevented its trapping after the removal of agonist. PB preferentially reduced the steady-state (IC(50) = 92.8 μM), rather than peak (IC(50) > 1 mM) component of responses evoked by glutamate and accelerated the onset of desensitization in a concentration-dependent manner. Miniature excitatory postsynaptic currents recorded from cultured hippocampal neurons, the time course of which is minimally influenced by desensitization, are not **inhibited** by PB. The sensitivity of **AMPAR**-mediated synaptic responses to **inhibition** by PB therefore depends on the contribution of desensitization to these events. Our results suggest that PB does not act as an open channel blocker of AMPARs. Rather, the sensitivity, use dependence, and trapping of **inhibition** by PB are determined by AMPARs desensitization.

L11 ANSWER 3 OF 21 MEDLINE on STN DUPLICATE 1
AN 2003252566 MEDLINE
DN PubMed ID: 12694947
TI AMPA receptors on developing medial septum/diagonal band neurons are sensitive to early postnatal binge-like ethanol exposure.
AU Hsiao Shu-Huei; Frye Gerald D
CS Department of Medical Pharmacology and Toxicology, Texas A&M University System Health Science Center, College of Medicine MS 1114, College Station, TX 77843-1114, USA.
NC AA 12386 (NIAAA)
SO Brain research. Developmental brain research, (2003 Apr 14) 142 (1) 89-99. Journal code: 8908639. ISSN: 0165-3806.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200307
ED Entered STN: 20030603
Last Updated on STN: 20030703
Entered Medline: 20030702
AB The impact of binge-like, early postnatal ethanol **treatment** on AMPA or kainate whole cell currents was examined in acutely isolated medial septum/diagonal band (MS/DB) neurons. AMPA (10 or 100 microM) current was **inhibited** by GYKI 52466, a selective AMPA receptor (**AMPAR**) **antagonist**, in all neurons isolated on postnatal day (PD) 5-8, PD 12-15 or PD 32-35. Cyclothiazide, a selective **inhibitor** of **AMPAR** desensitization, also effectively potentiated AMPA currents. This suggests that non-NMDA, ionotropic glutamate receptors on immature MS/DB neuron are predominantly AMPARs. Concentration-dependent kainate (10-1000 microM) application evoked nondesensitizing currents that exhibited an increase in the maximum response by the end of first postnatal month, consistent with developmental regulation of **AMPAR** function. Acute 3 s ethanol application (100 mM) consistently blunted AMPA- and kainate currents approximately 20-30% across age groups. **Inhibition** was sustained during continuous ethanol superfusion lasting 10-12 min without evidence of acute tolerance. Repeated oral intubation of **rat** pups with ethanol (5.25 g/kg/day on PD 4-9), which models third trimester human binge drinking, resulted in peak blood ethanol levels of approximately 350 mg/dl (measured 90 min after PD 6 dosing). AMPA or kainate currents were upregulated in neurons isolated on PD 32-35 by earlier ethanol intubation suggesting that binge-like intoxication augments developing **AMPAR** function. Despite this augmentation of **AMPAR** function, no significant changes were found in the sensitivity of AMPA currents to GYKI 52466, cyclothiazide or acute ethanol (100 mM) sensitivity or in the levels of GluR1/GluR2 subunit proteins from MS/DB tissue. These results indicate that non-NMDA ionotrophic glutamate receptors on immature MS/DB neurons, which are largely of the **AMPAR** subtype, are moderately sensitive to immediate **inhibition** by ethanol. Repeating this **inhibition** during early postnatal binge-like intoxication can augment normal development of **AMPAR** function.

L11 ANSWER 4 OF 21 MEDLINE on STN DUPLICATE 2
AN 2003098427 MEDLINE
DN PubMed ID: 12559123
TI Modification of the philanthotoxin-343 polyamine moiety results in different structure-activity profiles at muscle nicotinic ACh, NMDA and AMPA receptors.
AU Mellor I R; Brier T J; Pluteanu F; Stromgaard K; Saghyan A; Eldursi N; Brierley M J; Andersen K; Jaroszewski J W; Krogsgaard-Larsen P; Usherwood P N R
CS Division of Molecular Toxicology, School of Life and Environmental

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SO Neuropharmacology, (2003 Jan) 44 (1) 70-80.
Journal code: 0236217. ISSN: 0028-3908.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200304
ED Entered STN: 20030304
Last Updated on STN: 20030501
Entered Medline: 20030430
AB Voltage-dependent, non-competitive **inhibition** by philanthotoxin-343 (PhTX-343) analogues, with reduced charge or length, of nicotinic acetylcholine receptors (nAChR) of TE671 cells and ionotropic glutamate receptors (N-methyl-D-aspartate receptors (NMDAR) and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (**AMPAR**)) expressed in Xenopus oocytes from **rat** brain RNA was investigated. At nAChR, analogues with single amine-to-methylene or amine-to-ether substitutions had similar potencies to PhTX-343 (IC(50)=16.6 microM at -100 mV) whereas PhTX-(12), in which both secondary amino groups of PhTX-343 were replaced by methylenes, was more potent than PhTX-343 (IC(50)=0.93 microM at -100 mV). Truncated analogues of PhTX-343 were less potent. **Inhibition** by all analogues was voltage-dependent. PhTX-343 (IC(50)=2.01 microM at -80 mV) was the most potent **inhibitor** of NMDAR. At **AMPAR**, most analogues were equipotent with PhTX-343 (IC(50)=0.46 microM at -80 mV), apart from PhTX-83, which was more potent (IC(50)=0.032 microM at -80 mV), and PhTX-(12) and 4,9-dioxa-PhTX-(12), which were less potent (IC(50)s>300 microM at -80 mV). These studies show that PhTX-(12) is a selective nAChR **inhibitor** and PhTX-83 is a selective **AMPAR** **antagonist**.

L11 ANSWER 5 OF 21 MEDLINE on STN
AN 2003015991 MEDLINE
DN PubMed ID: 12522159
TI Chronic NMDA receptor blockade from birth delays the maturation of NMDA currents, but does not affect AMPA/kainate currents.
AU Colonnese Matthew T; Shi Jian; Constantine-Paton Martha
CS Department of Biology, Department of Brain and Cognitive Science, and McGovern Institute for Brain Research, Massachusetts Institute of Technology, Cambridge 02139, USA.
NC EY-06039 (NEI)
EY-104074 (NEI)
NS-32290 (NINDS)
SO Journal of neurophysiology, (2003 Jan) 89 (1) 57-68.
Journal code: 0375404. ISSN: 0022-3077.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20030111
Last Updated on STN: 20030327
Entered Medline: 20030326
AB The activity of the N-methyl-D-aspartate receptor (NR) regulates the composition of excitatory synapses and mediates multiple forms of synaptic and structural plasticity. In the superficial superior colliculus (sSC) of the **rat**, NR activity is essential for the full refinement of retinotopy during development. We have examined the NR's role in synaptic development by chronically **treating** the sSC from birth with the competitive **antagonist** (+/-)-2-amino-5-phosphonopentanoic acid (AP5) released by the slow-release polymer Elvax. Whole-cell voltage-clamp recordings were used to characterize excitatory postsynaptic

potentials (EPSCs) in slices from postnatal day (P)12-20 sSC. Chronic NR blockade reduced the ratio of AMPA/kainate receptor (**AMPAR**) to NR peak current amplitudes of both spontaneous (s)EPSCs and evoked EPSCs. Spontaneous NR current amplitude was increased following **treatment** , while spontaneous **AMPAR** currents were identical to those of controls, indicating that the ratio change was due to an increased NR current. Comparison of sEPSC frequency, **AMPAR** current rectification, and quantitative Western blots indicated that the characteristics of AMPARs at the synapse are normal following AP5 **treatment**. In the sSC, NR currents show a rapid decrease in decay time on P11 and previous studies in slices indicate this change results from a NR-mediated activation of the phosphatase calcineurin. Consistent with this in vitro finding, the down-regulation failed to occur in sSC chronically **treated** with AP5 in vivo. Together the present data show that NR function is necessary for subsequent NR current regulation in vivo, but it is not essential for the developmental expression of normal **AMPAR** currents.

L11 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:203006 BIOSIS
DN PREV200400203549
TI Developmental regulation of glutamate receptor subunits at the endbulb of Held - bushy cell synapse.
AU Bellingham, M. C. [Reprint Author]; Kerr, M. L. [Reprint Author]
CS Sch. of Biomed. Sci., Univ. of Queensland, Brisbane, Australia
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 676.17. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004
AB At endbulb-bushy cell synapses, alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (**AMPAR**)-mediated EPSCs increase in amplitude with age while N-methyl-D-aspartate receptor (NMDAR)-mediated EPSCs decrease in amplitude and decay time constant (tau decay). The functional characteristics of these receptors are dependent on subunit composition. For example, NMDARs containing NR2B subunits have high Ca2+ permeability, long tau decay, are typically more common in neonatal brains and are thought to play an important role in synapse development. Whole cell patch clamp recordings of single fibre-evoked EPSCs were made from bushy cells (n=59) in cochlear nucleus slices from postnatal day (P) 4-17 rats anesthetized with sodium pentobarbitone (20 mg/kg ip). Ifenprodil (10 microM), an NR2B subunit-selective NMDAR **antagonist**, reduced NMDAR EPSC amplitude to 24+-3% (mean+-SEM, n=13) of control in P4-8 rats, significantly greater than NMDAR EPSC reduction in P10-17 rats (40+-4% of control, n=13) suggesting a shift from NR2B to probably NR2A subunits. Pentobarbitone (100 microM), **inhibiting** AMPARs with GluR2 subunits, significantly reduced **AMPAR** EPSC amplitude in P4-6 rats to 51+-2% of control (n=4), to 73+-5% (n=3) at P8-11 and to 40+-14% (n=3) in P12-15 rats (P<0.05 between age groups). The intracellular polyamine spermine blocks Ca2+-permeable AMPARs lacking GluR2 subunits at positive voltages. After inclusion of spermine (100 microM) in the electrode solution, the mean rectification index (RI) of **AMPAR** EPSC I-Vs increased with age (P4-6, RI=1.2+-0.5 (n=5), P7-11, RI= 4.5+-0.5 (n=8), P12-15, RI= 5.6+-0.8 (n=10), suggesting that AMPARs in the two older groups are likely to lack GluR2 subunits and be more Ca2+ permeable. Altered Ca2+ permeability due to NMDAR and **AMPAR** subunit composition may be important in the development of the endbulb-bushy cell synapse.

L11 ANSWER 7 OF 21 MEDLINE on STN DUPLICATE 3

AN 2002455550 MEDLINE
DN PubMed ID: 12213255
TI Selective enhancement of AMPA receptor-mediated function in hippocampal CA1 neurons from chronic benzodiazepine-**treated** rats.
AU Van Sickle Bradley J; Tietz Elizabeth I
CS Department of Pharmacology, Medical College of Ohio, Toledo, OH 43614, USA.
NC F30-DA0604 (NIDA)
R01-DA0475 (NIDA)
SO Neuropharmacology, (2002 Jul) 43 (1) 11-27.
Journal code: 0236217. ISSN: 0028-3908.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200211
ED Entered STN: 20020906
Last Updated on STN: 20021212
Entered Medline: 20021121
AB Two days following one-week administration of the benzodiazepine, flurazepam (FZP), rats exhibit anticonvulsant tolerance in vivo, while reduced GABA(A) receptor-mediated **inhibition** and enhanced EPSP amplitude are present in CA1 pyramidal neurons in vitro. AMPA receptor (**AMPAR**)-mediated synaptic transmission in FZP-**treated** rats was examined using electrophysiological techniques in in vitro hippocampal slices. In CA1 pyramidal neurons from FZP-**treated** rats, the miniature excitatory postsynaptic current (mEPSC) amplitude was significantly increased (33%) without change in frequency, rise time or decay time. Moreover, mEPSC amplitude was not elevated in dentate granule neurons following 1-week FZP **treatment** or in CA1 pyramidal neurons following acute desalkyl-FZP **treatment**. Regulation of **AMPAR** number was assessed by quantitative autoradiography with the **AMPAR antagonist**, [(3)H]Ro48-8587. Specific binding was significantly increased in stratum pyramidale of hippocampal areas CA1 and CA2 and in proximal dendritic fields of CA1 pyramidal neurons. Regulation of **AMPAR** subunit proteins was examined using immunological techniques. Neither abundance nor distribution of GluR1-3 subunit proteins was different in the CA1 region following FZP **treatment** . These findings suggest that enhanced **AMPAR** currents, mediated at least in part by increased **AMPAR** number, may contribute to BZ anticonvulsant tolerance. Furthermore, these studies suggest an interaction between GABAergic and glutamatergic systems in the CA1 region which may provide novel therapeutic strategies for restoring BZ effectiveness.

L11 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:548526 BIOSIS
DN PREV200100548526
TI Complex mechanisms underlie long-term synaptic potentiation (LTP) of Aplysia sensorimotor connections induced by nerve shock.
AU Liao, X. [Reprint author]; Walters, E. T. [Reprint author]
CS Dept Integrative Biology Pharmacology, Univ Texas, Houston, TX, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 1703. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002
AB Under some conditions, LTP of synapses between Aplysia sensory neurons (SNs) and motor neurons (MNs) depends upon postsynaptic activation of an

NMDA-like receptor (NMDAR) and influx of calcium ions. Intense, high-frequency stimulation of peripheral nerves produces powerful LTP and should maximize the activation of NMDARs on MNs; but it should also release neuromodulators such as 5-HT that can produce long-lasting heterosynaptic facilitation and activity-dependent heterosynaptic facilitation by presynaptic actions. We found that the NMDAR blocker APV (100-300muM) failed to reduce LTP in tail MNs induced by stimulating nerve p9. The **AMPAR** blocker CNQX alone (75muM) or joint application of APV (100-300muM) and CNQX (10-75muM) also had no apparent effect on LTP 60-90 min after tetanus, but did reduce short-term potentiation (STP) at 15-30 min. Injection of BAPTA into tail MNs had the opposite effect, reducing LTP at 90-120 min without affecting STP. On the other hand, BAPTA infused into abdominal ganglion MNs failed to reduce LTP from siphon nerve stimulation. A 5-HT **antagonist**, methiothepin (200muM), did not significantly reduce STP or LTP. These results indicate that STP and LTP induced by peripheral nerve shock involve different mechanisms, and suggest that multiple processes contribute to LTP in these synapses. Currently we are investigating possible effects on LTP of presynaptic injection of PKI and other protein kinase **inhibitors** into abdominal ganglion SNs, and of combined application of different blockers.

L11 ANSWER 9 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:532942 BIOSIS
DN PREV200100532942
TI Kainate receptors on CA1 pyramidal cells **inhibit** calcium-dependent potassium current via PKC.
AU Melyan, Z. [Reprint author]; Lancaster, B.; Wheal, H. V. [Reprint author]
CS Centre for Neuroscience, University of Southampton, Southampton, UK
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1315. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
AB CA1 pyramidal cells contain mRNA for the GluR6 and KA2 subunits of glutamate receptor and respond to kainic acid (KA) and domoate application with inward currents. Despite the presence of these functional KA receptors, they make no ionotropic contribution to EPSPs in these cells. We tested the alternative possibility that these receptors have a metabotropic function in CA1 pyramidal cells. KA is known to **inhibit** a slow Ca2+-dependent K+ current (IsAHP) that follows brief depolarization. Bath application of KA caused concentration-dependent **inhibition** of IsAHP reaching a plateau of 34+-11% at 100 nM (n=6, IC50 apprx15 nM). This action was not accompanied by inward current and persisted in the presence of TTX/TEA (n=8), suggesting a direct action. KA **inhibition** of IsAHP was blocked by prior application of 20 muM CNQX (n=8), but not by **AMPAR**-preferring **antagonist** GYKI52466 (100 muM, n=5). Application of CNQX following KA did not relieve the long-lasting **inhibition**. Thus a second messenger, rather than persistent receptor activation, is likely to underlie the **inhibition**. KA action was mimicked by 200 nM domoate (51+-6% **inhibition**, n=7) but not by fluoro-willardine (300 nM, n=7) or the GluR5 subunit agonist ATPA (2 muM, n=5). These data are consistent with an action of KA via the GluR6 receptor subtype. As reported for presynaptic effects of KA, we found that preincubation of slices with the PKC **inhibitor** calphostin C (1 muM) blocked the action of KA (n=10). Subsequent application of noradrenaline (10 muM, n=4) blocked IsAHP, demonstrating that PKA-dependent **inhibition** remained intact.

L11 ANSWER 10 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:526984 BIOSIS
DN PREV200100526984
TI AMPA receptor activation **inhibits** GABA release from cerebellar interneurons through G protein-coupled mechanisms.
AU Satake, S. [Reprint author]; Murakoshi, T.; Konishi, S. [Reprint author]
CS Mitsubishi Kasei Inst. of Life Sci., and CREST, JST, Tokyo, Japan
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1305. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
AB We have reported that the climbing fiber (CF) transmitter **inhibits** GABA release from cerebellar interneurons via activation of AMPA-type glutamate receptors (AMPARs). To further explore the molecular mechanisms underlying the CF-induced disinhibition, we examined actions of AMPA on the GABAergic transmission at interneuron-Purkinje cell (PC) synapses in **rat** cerebellar thin slices. AMPA (0.5 muM) reduced the amplitude of stimulation-evoked **inhibitory** postsynaptic currents recorded from PCs. Pretreatment with N-ethylmaleimide (NEM, 250 muM, 5-10 min) markedly attenuated the AMPA-induced disinhibition, suggesting that Gi/o-coupled metabotropic pathways contribute to the downstream of **AMPAR** activation. Among intracellular signaling modulators tested, the calcineurin **inhibitor** FK506 (50 muM) and the phorbol ester PDBu (0.5 muM) significantly reduced the **AMPAR**-mediated disinhibition. However, forskolin (20 muM) and H-7 (30 muM) did not affect significantly the AMPA-induced actions, excluding involvements of Gi/o-mediated adenylate cyclase **inhibition** and subsequent downregulation of protein kinase A. The CF-induced disinhibition was also suppressed by NEM but not altered by FK506 and PDBu. BAPTA (40 mM) infusion into recorded PCs and the CB1 cannabinoid receptor **antagonist** AM251 (2 muM) superfusion did not cause any discernible changes in the CF-induced disinhibition. These observations suggest that neither depolarization-induced suppression of **inhibition** (DSI) nor activation of CB1 receptors in presynaptic terminals take part into the CF- and **AMPAR**-mediated **inhibition** of GABAergic transmission at cerebellar interneuron-PC synapses.

L11 ANSWER 11 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:532860 BIOSIS
DN PREV200100532860
TI mGluR5 enhances NMDA-mediated excitatory synaptic transmission in CA1 neurons via an IP3R-CA2+-PKC-PYK2-SRC cascade.
AU Kotecha, S. A. [Reprint author]; Roder, J. C. [Reprint author]; Orser, B. A. [Reprint author]; MacDonald, J. F. [Reprint author]
CS Dept Physiol, U. Toronto, Toronto, ON, Canada
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1294. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
AB Glutamate mediates excitatory synaptic transmission in the CNS by activating ionotropic (NMDA and AMPA) and metabotropic glutamate receptors (mGluR). In the CA1 region the primary group I mGluR is mGluR5 and its

activation, along with NMDARs, are required for the onset of longterm potentiation (LTP) - a model of learning and memory. Paradoxically, these receptors are also implicated in neuronal toxicity. However, the mechanism by which mGluR5 modulates NMDARs is poorly understood Using acutely isolated CA1 pyramidal neurons we determined that CHPG (mGluR5 agonist) enhanced NMDAR currents. This effect was attenuated with co-applications of MPEP (mGluR5 **antagonist**) and was absent in mGluR5 KO mice. The enhancement was dependent upon co-incident NMDAR gating as co-applications of reversible, open-channel blockers during CHPG failed to enhance NMDAR currents during prolonged wash. The mGluR5-effect was blocked by selective **inhibitors** to PKC and the tyrosine kinases Pyk2 and Src. Moreover, administration of a PKC activator and inclusion of recombinant Pyk2 and Src in the patch electrode occluded the CHPG-effect. Buffering of (Ca2+)i and inclusion of thapsigargin (IP3R activator) blocked and occluded, respectively, the mGluR5 effect. Thapsigargin enhanced NMDAR currents and **inhibitors** to PKC, Pyk2 and Src blocked this effect. Recordings from cultured hippocampal neurons revealed that CHPG potentiated the NMDAR, but not **AMPAR**, component of mEPSCs. **Inhibitors** to mGluR5 and Pyk2 blocked this enhancement. Given that mGluR5, PKC, Pyk2 and Src are implicated in LTP, these results may provide a unifying model in which NMDARs act as a molecular switch for the induction of CA1-LTP.

L11 ANSWER 12 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:532794 BIOSIS
DN PREV200100532794
TI Spontaneous synchronized calcium oscillations in neocortical neurons in the presence of physiological (MG2+): Involvement of AMPA/kainate receptors and metabotropic glutamate receptors.
AU Dravid, S. M. [Reprint author]; Murray, T. F. [Reprint author]
CS Department of Physiology and Pharmacology, College of Vet. Medicine, UGA, Athens, GA, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1269. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
AB Primary cultures of neocortical neurons and other neuronal cell types have been shown to exhibit spontaneous calcium oscillations under zero or low extracellular (Mg2+). We find that murine neocortical neurons cultured for 9-13 days produce calcium oscillations in the presence of physiological (Mg2+). Intracellular calcium ((Ca2+)i) monitoring was done in fluo-3 loaded neocortical neurons using a fluorescent laser imaging plate reader (FLIPR). Calcium oscillations were action potential mediated inasmuch as tetrodotoxin eliminated their occurrence. The finding that NBQX suppressed these oscillations indicates that they are triggered by AMPA/kainate receptors. Moreover cyclothiazide, an **inhibitor** of **AMPAR** desensitization, enhanced the frequency of oscillations. In contrast, concanavalin A, an **inhibitor** of kainate receptor desensitization, had no effect. NMDA receptors do not appear to be involved in generation of calcium oscillations due to their insensitivity to MK-801 (100nM). S-4-carboxyphenylglycine, an **antagonist** of group I metabotropic glutamate receptor (mGluR), reduced the amplitude of oscillations suggesting integration of multiple pathways in the regulation of these oscillations. Depletion of (Ca2+)i stores with thapsigargin also reduced the amplitude of the oscillations indicating a contribution of (Ca2+)i stores in this phenomenon. The present study indicates that spontaneous calcium oscillations in neocortical cultures are primarily initiated by AMPA receptors and involve mobilization of intracellular

calcium stores following activation of mGluR.

L11 ANSWER 13 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:551707 BIOSIS
DN PREV200100551707
TI Glutamate-mediated extrasynaptic **inhibition** in the **rat** olfactory bulb.
AU Isaacson, J. S. [Reprint author]; Murphy, G. J. [Reprint author]
CS Neuroscience, UCSD, La Jolla, CA, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1210. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
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LA English
ED Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002
AB NMDA receptors (NMDARs) mediate excitatory synaptic transmission and Ca2+ influx via NMDARs underlies synaptic plasticity in the CNS. Here we show that synaptically-released glutamate evokes a slow, NMDAR-mediated **inhibitory** postsynaptic current (IPSC) in olfactory bulb granule cells. **Rat** olfactory bulb slices were superfused (31-33 C) with a Ringer solution containing picrotoxin (100 muM). Granule cells were patch-clamped with electrodes containing a K+-based internal solution. Synaptic transmission was evoked via a stimulating electrode in the granule cell layer to activate mitral cell axons. Brief stimulus trains (50 Hz, 20 pulses) evoked fast AMPA receptor (**AMPAR**)-mediated excitatory synaptic currents (EPSCs) at negative holding potentials. Membrane depolarization (Vh=-10 mV) revealed an inward NMDAR EPSC that was curtailed by a slow outward IPSC. This slow IPSC was unaffected by the **AMPAR antagonist** NBQX (20 muM) but was abolished by the NMDAR **antagonist** APV (50 muM). The BK channel antagonists iberiotoxin (200 nM) and paxilline (10 muM) blocked the slow IPSC and revealed an underlying NMDAR EPSC. Briefer trains (1-5 pulses) evoked inward NMDAR-mediated currents but failed to produce a slow IPSC. However, under these conditions application of the glutamate uptake blockers DHK (100 muM) or trans-PDC (200 muM) revealed a slow BK channel-mediated IPSC. Similar results were obtained by lowering the temperature (25 C) to slow glutamate uptake. Together, these results indicate that glutamate diffuses to extrasynaptic NMDARs to generate the slow IPSC. These findings suggest that glutamate spillover governs extrasynaptic **inhibition** via the coupling of NMDAR-mediated Ca2+ influx to BK channel activation.

L11 ANSWER 14 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4
AN 2001:320467 BIOSIS
DN PREV200100320467
TI 6-Hydroxykynurenic acid and kynurenic acid differently antagonise AMPA and NMDA receptors in hippocampal neurones.
AU Weber, Marco; Dietrich, Dirk; Graesel, Ines; Reuter, Gerhard; Seifert, Gerald; Steinhaeuser, Christian [Reprint author]
CS Experimental Neurobiology, Neurosurgery, Bonn University, Sigmund-Freud-Str. 25, 53105, Bonn, Germany
Christian.Steinhaeuser@ukb.uni-bonn.de
SO Journal of Neurochemistry, (May, 2001) Vol. 77, No. 4, pp. 1108-1115. print.
CODEN: JONRA9. ISSN: 0022-3042.
DT Article
LA English
ED Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002

AB 6-Hydroxykynurenic acid (6-HKA), a derivative of kynurenic acid (KYNA) extracted from Ginkgo biloba leaves, was tested for its putative glutamate receptor (GluR) antagonism in comparison to the scaffold substance. The patch-clamp method together with fast-application techniques were used to estimate **inhibition** by 6-HKA and KYNA of agonist binding at NMDA and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors (NMDARs and AMPARs) of CA1 pyramidal neurones. 6-Hydroxykynurenic acid proved to be a low-affinity **antagonist**. When comparing with KYNA, 6-HKA was less potent at NMDARs (IC50 = 136 versus 59 muM), but showed a higher affinity to AMPARs (KB = 22 versus 172 muM). The replacement of 6-HKA and KYNA by glutamate was investigated on outside-out patches. Both antagonists competitively **inhibited** **AMPAR** responses and displayed fast unbinding kinetics, but the derivative was significantly slower displaced than KYNA (tau = 1.63 versus 1.22 ms). Our findings demonstrate that 6-hydroxylation considerably changes the pharmacological profile of KYNA. Among the 6-derivatives of KYNA, 6-HKA shows the highest affinity to AMPARs. Despite its relatively low lipophily, these properties might be of clinical relevance under conditions that compromise the integrity of the blood-brain barrier. Furthermore, 6-HKA should be a useful tool to analyse glutamate-mediated synaptic responses.

L11 ANSWER 15 OF 21 MEDLINE on STN
AN 2001185547 MEDLINE
DN PubMed ID: 11277576
TI Extension of glial processes by activation of Ca2+-permeable AMPA receptor channels.
AU Ishiuchi S; Tsuzuki K; Yamada N; Okado H; Miwa A; Kuromi H; Yokoo H; Nakazato Y; Sasaki T; Ozawa S
CS Department of Neurosurgery, Gunma University School of Medicine, Maebashi, Japan.
SO Neuroreport, (2001 Mar 26) 12 (4) 745-8.
Journal code: 9100935. ISSN: 0959-4965.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010806
Last Updated on STN: 20010806
Entered Medline: 20010802
AB AMPA type-glutamate receptor channels (AMPARs) assembled without the GluR2 (GluR-B) subunit are characterized by high Ca2+ permeability, and are expressed abundantly in cerebellar Bergmann glial cells. Here we show that the morphology of cultured Bergmann glia-like fusiform cells derived from the **rat** cerebellum was changed by manipulating expression of Ca2+-permeable AMPARs using adenoviral vector-mediated gene transfer. Converting endogenous Ca2+-permeable AMPARs into Ca2+-impermeable channels by viral-mediated transfer of GluR2 gene induced retraction of glial processes. In contrast, overexpression of Ca2+-permeable AMPARs markedly elongated glial processes. The process extension was blocked by 2,3-Dihydroxy-6-nitro-7-sulfamoylbenzo(F)quinoxaline (NBQX), a specific **antagonist** of **AMPAR**. These results indicate that glutamate regulates the morphology of glial processes by activating Ca2+-permeable AMPARs.

L11 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:487249 BIOSIS
DN PREV200100487249
TI Synaptic activation of extrasynaptic NMDA receptors on ganglion cells in **rat** retina.
AU Chen, S. [Reprint author]; Diamond, J. S. [Reprint author]
CS Synaptic Physiology Unit, NINDS/NIH, Bethesda, MD, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 407. print.

Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Oct 2001
Last Updated on STN: 23 Feb 2002
AB Excitatory synaptic inputs onto ganglion cells were studied with patch recordings in acute slices of **rat** retina. Electrically-evoked, excitatory postsynaptic currents (EPSCs) exhibited two components: a fast component with an ohmic conductance and a slow, voltage-dependent component. The fast component was blocked by the AMPA receptor (**AMPAR**) **antagonist** DNQX and the slow component was blocked by the NMDA receptor (NMDAR) **antagonist** CPP. Miniature EPSCs (mEPSCs), reflecting the postsynaptic response to a single quantum of transmitter, exhibited only one component, which was completely blocked by DNQX; no NMDA component was observed in mEPSCs, even when recording at -80 mV in Mg2+-free solutions. The results suggest that mEPSCs in **rat** ganglion cells are mediated solely by AMPARs, while, electrically-evoked EPSCs reflect concomitant activation of NMDARs and AMPARs. We also used low-affinity competitive antagonists to estimate the glutamate concentration sensed by both receptor types. The **inhibition** of EPSCs by low-affinity antagonists was compared to **antagonist inhibition** of responses elicited by 1 mM glutamate in outside-out patches. The low-affinity NMDAR **antagonist**, L-AP5, exerted a greater relative block than the low-affinity **AMPAR antagonist**, gamma-DGG, suggesting that AMPARs encounter a higher glutamate concentration than NMDARs during an EPSC. The glutamate uptake **inhibitor** TBOA enhanced NMDAR mediated EPSCs, and caused an NMDAR-mediated component to emerge in mEPSCs. We conclude that NMDARs on ganglion cells are located extrasynaptically and are activated only when glutamate is released simultaneously from multiple sites.

L11 ANSWER 17 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2000222600 EMBASE
TI Kainate receptor-mediated synaptic currents in cerebellar Golgi cells are not shaped by diffusion of glutamate.
AU Bureau I.; Dieudonn S.; Coussen F.; Mulle C.
CS C. Mulle, Ctr. Natl. de la Rech. Scientifique, UMR 5091, Institut Francois Magendie, Bordeaux 33077, France. mulle@u-bordeaux2.fr
SO Proceedings of the National Academy of Sciences of the United States of America, (6 Jun 2000) 97/12 (6838-6843).
Refs: 43
ISSN: 0027-8424 CODEN: PNASA6
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
LA English
SL English
AB We report the presence of kainate receptors (KARs) in cerebellar Golgi cells of wild-type but not GluR6-deficient mice. Parallel fiber stimulation activates KAR-mediated synaptic currents [KAR-excitatory postsynaptic currents (EPSCs)] of small amplitude. KAR-EPSCs greatly differ from synaptic currents mediated by α-amino-3-hydroxy-5-methylisoxazole-4-propionate (AMPA) receptors (**AMPAR**-EPSCs) at the same synapse. KAR-EPSCs display slow rise and decay time and summate in response to a train of stimulations. By using PDA, a low-affinity competitive **antagonist** and agents that modify the clearance of glutamate, we show that these properties cannot be explained by diffusion of glutamate outside of the synaptic cleft and activation of extrasynaptic KARs. These data suggest that the slow kinetic of KAR-EPSCs is due to

intrinsic properties of KARs being localized at postsynaptic sites. The contrasting properties of KAR- and **AMPAR**-EPSCs in terms of kinetics and summation offer the possibility for a glutamatergic synapse to integrate excitatory inputs over two different time scales.

L11 ANSWER 18 OF 21 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:80993 BIOSIS
DN PREV200100080993
TI Mechanisms governing dendritic GABA release from granule cells in the **rat** olfactory bulb.
AU Isaacson, J. S. [Reprint author]
CS UCSD, La Jolla, CA, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-519.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Feb 2001
Last Updated on STN: 12 Feb 2002
AB Glutamate release from mitral cell dendrites excites the dendrites of granule cells, which mediate GABAergic dendrodendritic **inhibition** (DDI) back onto mitral cells. NMDA receptors (NMDARs) play a critical role in DDI (Isaacson and Strowbridge, 1998). It has been suggested that Ca2+ influx through NMDARs triggers GABA release from granule dendrites (Chen et al., 2000). Alternatively, NMDAR-mediated depolarization may recruit voltage-gated Ca2+ channels (VGCCs) that govern release. To address this, we examined whether DDI has an absolute requirement for NMDARs. We studied DDI in bulb slices (300 mum) superfused with solution containing TTX (1 muM) and 1.3 mM Mg2+. Mitral cells were recorded with a CsCl-based internal solution at -70 mV. DDI was evoked by 50 ms voltage steps to 0 mV to activate VGCCs in mitral dendrites. Addition of the NMDAR antagonists APV (100 muM) and MK-801 (40 muM) greatly reduced DDI, confirming that NMDA receptors play a dominant role in triggering GABA release. One possibility is that the slow kinetics of NMDARs are important for bringing granule cells to threshold for activating VGCCs that govern GABA release. We studied the actions of cyclothiazide, a drug that slows the kinetics of AMPA receptors (AMPARs). In the presence of APV/MK-801, cyclothiazide (200 muM) restored DDI and this response was abolished by the **AMPAR antagonist** NBQX (10 muM).
Increasing granule cell excitability with the K+ channel blockers 4-AP (200 muM) and TEA (2 mM) also rescued DDI in APV/MK-801 and this action was abolished by NBQX. These results indicate that GABA release from granule spines can be driven entirely by AMPARs. These findings are consistent with a role for VGCCs in mediating granule dendrite GABA release.

L11 ANSWER 19 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 1998222498 EMBASE
TI Potentiation of GABAergic synaptic transmission by AMPA receptors in mouse cerebellar stellate cells: Changes during development.
AU Bureau I.; Mulle C.
CS C. Mulle, CNRS UMR 5541, Universite Victor Segalen-Bordeaux 2, 146 rue Leo-Saignat, 33076 Bordeaux, France. mulle@hippocrate.u-bordeaux2.fr
SO Journal of Physiology, (15 Jun 1998) 509/3 (817-831).
Refs: 44
ISSN: 0022-3751 CODEN: JPHYA7
CY United Kingdom
DT Journal; Article
FS 002 Physiology
008 Neurology and Neurosurgery

LA English
SL English
AB 1. The effects of low concentrations of domoate, an agonist at both α-amino-3-hydroxy-5-methylisoxazole-4-propionate and kainate receptors (AMPARs and KARs, respectively), were investigated in stellate cells in slices of mouse cerebellum at two developmental stages (postnatal day (PN) 11-13 and PN21-25). 2. Low concentrations of domoate enhanced the frequency of miniature IPSCs (mIPSCs) recorded in the presence of tetrodotoxin (TTX) at PN11-13 but not at PN21-25. 3. The effects of low concentrations of domoate on synaptic activity were probably mediated by the activation of AMPARs and not KARs, since they were blocked by GYKI 53655 (LY300168), a selective **AMPAR antagonist**. 4. Domoate increased mIPSC frequency in part by activation of presynaptic voltage-dependent Ca2+ channels since potentiation was reduced by 60% in the presence of Cd2+. AMPARs in stellate cells were found to be permeable to Ca2+. The residual potentiation in the presence of Cd2+ could thus be due to a direct entry of Ca2+ through **AMPAR** channels. 5. In the presence of TTX, potentiation of synaptic activity by focal application of domoate was not restricted to the region of the cell body but was observed within distances of 120 μm. These experiments also revealed a strong spatial correlation between the location of the presynaptic effects of domoate and the activation of postsynaptic AMPARs. 6. Our data show a developmentally regulated presynaptic potentiation of synaptic transmission between cerebellar interneurones mediated by AMPARs. We discuss the possibility that the developmental switch could be due to a shift in the localization of AMPARs from the axonal to the somato-dendritic compartment.

L11 ANSWER 20 OF 21 MEDLINE on STN
AN 97138928 MEDLINE
DN PubMed ID: 8985912
TI Enhanced NMDAR-dependent epileptiform activity is controlled by oxidizing agents in a chronic model of temporal lobe epilepsy.
AU Hirsch J C; Quesada O; Esclapez M; Gozlan H; Ben-Ari Y; Bernard C L
CS Institut National de la Sante et de la Recherche Medicale U29, Hopital de Port Royal, Paris, France.
SO Journal of neurophysiology, (1996 Dec) 76 (6) 4185-9.
Journal code: 0375404. ISSN: 0022-3077.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199704
ED Entered STN: 19970414
Last Updated on STN: 19970414
Entered Medline: 19970402
AB 1. Graded N-methyl-D-aspartate receptor (NMDAR)-dependent epileptiform discharges were recorded from ex vivo hippocampal slices obtained from rats injected a week earlier with an intracerebroventricular dose of kainic acid. Intracellular recordings from pyramidal cells of the CA1 area showed that glutamate NMDAR actively participated in synaptic transmission, even at resting membrane potential. When NMDAR were pharmacologically isolated, graded burst discharges could still be evoked. 2. The oxidizing reagent 5,5'-dithiobis(2-nitrobenzoic acid) (DTNB, 200 microM, 15 min) suppressed the late part of the epileptiform burst that did not recover after wash but could be reinstated by the reducing agent tris (2-carboxyethyl) phosphine (TCEP, 200 microM, 15 min) and again abolished with the NMDA **antagonist** D-2-amino-5-phosphonovaleric acid (D-APV). 3. Pharmacologically isolated NMDAR-mediated responses were decreased by DTNB (56 +/- 10%, mean +/- SD, n = 6), an effect reversed by TCEP. 4. When only the fast glutamateric synaptic component was blocked, NMDA-dependent excitatory postsynaptic potentials (EPSPs) could be evoked despite the presence of underlying fast and slow **inhibitory** postsynaptic potentials (IPSPs). DTNB decreased EPSPs to 48 +/- 12% (n =

5) of control. 5. Since a decrease of the NMDAR-mediated response by +/- 50% is sufficient to suppress the late part of the burst, we suggest that epileptiform activity can be controlled by manipulation of the redox sites of NMDAR. Our observations raise the possibility of developing new anticonvulsant drugs that would spare alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid-R (**AMPAR**)-mediated synaptic responses and decrease NMDAR-mediated synaptic transmission without blocking it completely.

L11 ANSWER 21 OF 21 MEDLINE on STN DUPLICATE 5
AN 95198083 MEDLINE
DN PubMed ID: 7891148
TI Tetanically induced LTP involves a similar increase in the AMPA and NMDA receptor components of the excitatory postsynaptic current: investigations of the involvement of mGlu receptors.
AU O'Connor J J; Rowan M J; Anwyl R
CS Department of Pharmacology and Therapeutics, Trinity College, Dublin, Ireland.
SO Journal of neuroscience : official journal of the Society for Neuroscience, (1995 Mar) 15 (3 Pt 1) 2013-20.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199504
ED Entered STN: 19950427
Last Updated on STN: 19950427
Entered Medline: 19950420
AB Whole-cell patch-clamp recordings of evoked excitatory postsynaptic currents (EPSCs) were made from granule cells of the **rat** dentate gyrus in vitro. Tetanic stimulation in control media evoked a statistically identical long-term potentiation (LTP) of both the AMPA and NMDA receptor-mediated components of the dual component EPSC (AM-PAR and NMDAR EPSCs), as shown by a similar percentage increase in both components when measured at a holding potential of -30 mV, and also by an identical time course of the pre- and post-LTP induced EPSC at -30 mV and -70 mV. Application of the selective metabotropic glutamate receptor (mGluR) agonist 1S,3R-ACPD induced a transient depression followed by a rapid onset LTP of both the **AMPAR** and the NMDAR components of the dual component EPSC. The ACPD- and tetanically induced LTP of the **AMPAR** EPSC was NMDAR dependent, being abolished by the NMDAR **antagonist** AP5. Tetanic stimulation, and application of ACPD, also induced a relatively rapid onset LTP of the pharmacologically isolated NMDAR EPSC. Such tetanically and ACPD-induced LTP of the isolated NMDAR EPSC was also dependent on NMDAR activation, being strongly **inhibited** by AP5. The tetanically and the ACPD-induced LTP of the NMDAR EPSC were dependent on protein kinase C (PKC) stimulation, being strongly **inhibited** by the PKC **inhibitor** PKCI (19-31).
The studies suggest that coactivation of the mGluR and NMDAR are required for induction of LTP of both the **AMPAR**- and NMDAR-mediated synaptic transmission. Moreover, LTP of the NMDAR-mediated synaptic transmission appears to be dependent on coincident activation of the NMDAR and mGluR.

=> d his

(FILE 'HOME' ENTERED AT 08:47:04 ON 10 JUN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:47:18 ON 10 JUN 2004
L1 57 S (AMPA ADJ RECEPTOR OR AMPAR) AND TREAT?
L2 43 S L1 AND (CLINICAL OR ANIMAL OR IN ADJ VIVO OR MAMMAL OR MOUSE
L3 28 DUP REM L2 (15 DUPLICATES REMOVED)

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L4            29 S L1 NOT L3
L5            19 DUP REM L4 (10 DUPLICATES REMOVED)
L6           164 S (AMPA ADJ RECEPTOR OR AMPAR) AND INHIBIT?
L7            17 S L6 AND TREAT?
L8            13 DUP REM L7 (4 DUPLICATES REMOVED)
L9            38 S L6 AND (ANTAGONIST OR QUINAZOLIN?)
L10           30 S L9 AND (TREAT? OR RAT OR MONKEY OR ANIMAL OR (IN ADJ VIVO))
L11           21 DUP REM L10 (9 DUPLICATES REMOVED)

=> l9 not l10
L9 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l9 not l10
L12            8 L9 NOT L10

=> dup rem
ENTER L# LIST OR (END):l12
PROCESSING COMPLETED FOR L12
L13            7 DUP REM L12 (1 DUPLICATE REMOVED)

=> d bib abs l13 1-7

L13  ANSWER 1 OF 7     MEDLINE on STN                    DUPLICATE 1
AN   2004109145     MEDLINE
DN   PubMed ID: 14999062
TI   Two Loci of expression for long-term depression at hippocampal mossy
     fiber-interneuron synapses.
AU   Lei Saobo; McBain Chris J
CS   Laboratory of Cellular and Synaptic Neurophysiology, National Institute of
     Child Health and Human Development, National Institutes of Health,
     Bethesda, Maryland 20892-4495, USA.
SO   Journal of neuroscience : official journal of the Society for
     Neuroscience, (2004 Mar 3) 24 (9) 2112-21.
     Journal code: 8102140. ISSN: 1529-2401.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200405
ED   Entered STN: 20040305
     Last Updated on STN: 20040515
     Entered Medline: 20040514
AB   Two distinct forms of long-term depression (LTD) exist at mossy fiber
     synapses between dentate gyrus granule cells and hippocampal CA3 stratum
     lucidum interneurons.  Although induction of each form of LTD requires an
     elevation of postsynaptic intracellular Ca2+, at Ca2+-impermeable AMPA
     receptor (CI-AMPAR) synapses, induction is NMDA receptor (NMDAR)
     dependent, whereas LTD at Ca2+-permeable AMPA receptor (CP-AMPAR
     ) synapses is NMDAR independent.  However, the expression locus of either
     form of LTD is not known.  Using a number of criteria, including the
     coefficient of variation, paired-pulse ratio, AMPA-NMDA receptor activity,
     and the low-affinity AMPAR antagonist
     gamma-D-glutamyl-glycine, we demonstrate that LTD expression at CP-
     AMPAR synapses is presynaptic and results from reduced transmitter
     release, whereas LTD expression at CI-AMPAR synapses is
     postsynaptic.  The N-ethylmaleimide-sensitive fusion protein-AP2-clathrin
     adaptor protein 2 inhibitory peptide pep2m occluded LTD
     expression at CI-AMPAR synapses but not at CP-AMPAR
     synapses, confirming that CI-AMPAR LTD involves postsynaptic
     AMPAR trafficking.  Thus, mossy fiber innervation of CA3 stratum
     lucidum interneurons occurs via two parallel systems targeted to either
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Ca2+-permeable or Ca2+-impermeable AMPA receptors, each with a distinct expression locus for long-term synaptic plasticity.

L13 ANSWER 2 OF 7 MEDLINE on STN
AN 2003071589 MEDLINE
DN PubMed ID: 12581776
TI Binding modes of noncompetitive AMPA antagonists: a computational approach.
AU De Luca Laura; Macchiarulo Antonio; Costantino Gabriele; Barreca Maria Letizia; Gitto Rosaria; Chimirri Alba; Pellicciari Roberto
CS Dipartimento Farmaco-Chimico, Universita di Messina, Viale Annunziata 98168, Messina, Italy.
SO Farmaco (Societa chimica italiana : 1989), (2003 Feb) 58 (2) 107-13. Journal code: 8912641. ISSN: 0014-827X.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200309
ED Entered STN: 20030214
Last Updated on STN: 20030903
Entered Medline: 20030902
AB The activity of functional AMPA receptors (AMPARs) is modulated by noncompetitive antagonists. So far, no information about the molecular mechanism of action and the localization of the binding pocket(s) is available. We speculated that the leucine/isoleucine/valine binding protein (LIVBP)-like domain of **AMPAR**, localized at the extracellular N-terminus of the receptor, might be involved in the binding of noncompetitive antagonists and we tested this hypothesis through a computational approach involving the comparison with NMDA and metabotropic glutamate receptors and the generation of a 3D homology model of the LIVBP-like domain of **AMPAR**. The results suggest that the interdomain cleft of the LIVBP-like domain of **AMPAR** may contain the noncompetitive **antagonist** binding pocket.
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L13 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:198230 BIOSIS
DN PREV200400198789
TI Gene knockout study of glycine transporter I.
AU Coyle, J.; Tsai, G.; Bergeron, R. [Reprint Author]; Martina, M. [Reprint Author]; Berger-Sweeney, J.
CS Psychiatry, Ottawa Hlth. Res. Inst., Ottawa, ON, Canada
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 373.15. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004
AB NMDA receptor (NMDAR) activation requires the binding of both glutamate to its recognition site and glycine or D-serine to the glycine modulatory site (GMS). The glycine transporter (GlyT1) regulates the glycine level at the NMDAR. To understand better its role in NMDAR, we generated mice lacking a functional GlyT1 by homologous recombination to delete exons 2 and 3, which are common to all the isoforms GlyT1A-D. As GlyT1-/-died immediately after birth, we studied the GlyT1+/-heterozygotes. Using whole-cell patch-clamp recording of CA1 pyramidal cells in acute hippocampal slices obtained from mice of 3 months of age, we found that the application of exogenous glycine did not increase the amplitude of the NMDAR current in GlyT1+/-, suggesting that the GMS may be saturated. Comparing GlyT1+/-to WT, the decay time constants of the NMDAR currents

were faster, the **inhibitory** effect of ifenprodil, a specific NR2B **antagonist**, on the amplitude of the NMDAR current was smaller, suggesting a different molecular composition of the NMDAR in the GlyT1+/-CA1. The frequency of the EPSCs was similar, while the decay time constant was slower. The mEPSCs quantal size and the **AMPAR** /NMDAR ratio were smaller but the TTX-sensitive spontaneous mEPSCs were larger, suggesting that Schaffer collaterals may have more synaptic contacts with individual CA1 pyramidal cells. Consistent with these findings, GlyT1+/-mice have better memory retention as tested in the Morris water maze. Our results suggest that the GlyT1+/-have a saturating level of glycine in the synaptic cleft due to an impairment in glycine buffering. This causes a hyper-function of the NMDARs that results in altered subunit expression, the number of AMPARs in the synapse, and abnormal synaptogenesis. These findings suggest that **inhibition** of GlyT1 may be a feasible target for the development of drugs for disorders related to hypofunction of NMDAR.

L13 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:294271 BIOSIS
DN PREV200300294271
TI NITRIC OXIDE ENHANCES CORTICAL FEEDBACK IN THE THALAMIC RETICULAR NUCLEUS.
AU Kurukulasuriya, N. C. [Reprint Author]; Alexander, G. M. [Reprint Author]; Godwin, D. W. [Reprint Author]
CS Department of Neurobiology and Anatomy, Neuroscience Program, Wake Forest University School of Medicine, Winston Salem, NC, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 352.20. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003
AB Terminals from the brain stem parabrachial region (PBR) containing brain nitric oxide synthase innervate both the LGN and TRN. We previously showed that nitric oxide (NO) released from the PBR enhances corticothalamic (CT) EPSPs in the LGN. Since TRN cells comprise a major target for CT input, the effect of NO on EPSPs in the TRN is of particular significance. We hypothesized that NO influences CT EPSPs in the TRN. We tested this with intracellular recordings in adult ferret thalamic slices. We elicited EPSPs in the TRN via a stimulating electrode (1muA, 0.1msec pulses) placed in the optic radiations. GABAA and GABAB IPSPs were blocked with 150muM bicuculline methiodide and 200muM 2-OH-saclofen, respectively. As with TC cells, CT EPSPs in hyperpolarized TRN cells were rapid, while a slower component appeared at depolarized potentials (n=24). Paired pulse facilitation of the CT EPSPs was apparent (n=10). The delayed EPSP in the TRN was shorter compared to those seen in the LGN. The rapid component was DNQX (30muM) sensitive and **AMPAR** mediated (n=3), while the delayed component was APV (150muM) sensitive and NMDAR mediated (n=3). Application of the NO donor S-nitroso-N-acetyl-DL (SNAP,2mM) selectively enhanced the NMDAR component of the TRN CT EPSP (n=5), sometimes transforming the EPSP into a burst.)Voltage isolation of the AMPA component revealed that NO did not significantly alter AMPA transmission (n=6). These findings suggest common themes in the way CT inputs are targeted by NO: NMDAR mediated transmission is enhanced by NO in both TC and TRN cells.

L13 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:532937 BIOSIS
DN PREV200100532937
TI Short-term plasticity at the retinogeniculate synapse.
AU Chen, C. [Reprint author]; Blitz, D. M.; Regehr, W. G.

CS Div of Neurosci, Children's Hospital, Harvard Med Sch, Boston, MA, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1314. print.
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DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Nov 2001
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AB Visual information, encoded in the firing patterns of retinal ganglion cells, is transmitted to the brain via the retinogeniculate synapse. To identify the mechanisms of short-term plasticity at this connection, we examined synaptic currents evoked by stimulation of single retinal ganglion cell axons in mouse lateral geniculate nucleus brain slices (p28-31, 24C). Paired-pulse plasticity of AMPA and NMDA receptor (**AMPAR** and NMDAR) components was studied. While both components depressed, at short interpulse intervals (isi) there were differences in the amplitude and duration of the plasticity. Maximal depression was 25% and 50% of control for the AMPA EPSC and NMDA EPSC, respectively. There was a rapid phase of recovery for both components, with a time constant of 120 ms for the **AMPAR** and 170 ms for the NMDAR. Both receptor types also had a slow phase of recovery from depression with a time constant of apprx2 sec. We found that cyclothiazide, at concentrations that **inhibit AMPAR** desensitization without affecting presynaptic release, relieves the fast component of **AMPAR** depression. Experiments with NMDAR antagonists with different kinetic properties indicate that at short isi saturation contributes to depression of the NMDA EPSC. When saturation of the NMDAR is relieved depression is similar to that of the **AMPAR** when desensitization is eliminated. These findings suggest that the slow phase of recovery reflects a presynaptic form of depression. The rapid phases of recovery, however, reflect **AMPAR** desensitization and NMDAR saturation. This is consistent with a synaptic structure containing many release sites in dose proximity.

L13 ANSWER 6 OF 7 MEDLINE on STN
AN 2001276300 MEDLINE
DN PubMed ID: 11359876
TI 6-Hydroxykynurenic acid and kynurenic acid differently antagonise AMPA and NMDA receptors in hippocampal neurones.
AU Weber M; Dietrich D; Grasel I; Reuter G; Seifert G; Steinhauser C
CS Experimental Neurobiology, Neurosurgery, Bonn University, Bonn, Germany
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SO Journal of neurochemistry, (2001 May) 77 (4) 1108-15.
Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200106
ED Entered STN: 20010625
Last Updated on STN: 20021211
Entered Medline: 20010621
AB 6-Hydroxykynurenic acid (6-HKA), a derivative of kynurenic acid (KYNA) extracted from Ginkgo biloba leaves, was tested for its putative glutamate receptor (GluR) antagonism in comparison to the scaffold substance. The patch-clamp method together with fast-application techniques were used to estimate **inhibition** by 6-HKA and KYNA of agonist binding at NMDA and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors (NMDARs and AMPARs) of CA1 pyramidal neurones. 6-Hydroxykynurenic acid proved to be a low-affinity **antagonist**. When comparing with KYNA, 6-HKA was less potent at NMDARs (IC(50) = 136

versus 59 microM), but showed a higher affinity to AMPARs (K(B) = 22 versus 172 microM). The replacement of 6-HKA and KYNA by glutamate was investigated on outside-out patches. Both antagonists competitively **inhibited AMPAR** responses and displayed fast unbinding kinetics, but the derivative was significantly slower displaced than KYNA (tau = 1.63 versus 1.22 ms). Our findings demonstrate that 6-hydroxylation considerably changes the pharmacological profile of KYNA. Among the 6-derivatives of KYNA, 6-HKA shows the highest affinity to AMPARS: Despite its relatively low lipophily, these properties might be of clinical relevance under conditions that compromise the integrity of the blood-brain barrier. Furthermore, 6-HKA should be a useful tool to analyse glutamate-mediated synaptic responses.

L13 ANSWER 7 OF 7 MEDLINE on STN
AN 1998261644 MEDLINE
DN PubMed ID: 9596802
TI Potentiation of GABAergic synaptic transmission by AMPA receptors in mouse cerebellar stellate cells: changes during development.
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CS CNRS UMR 5541, Universite Victor Segalen-Bordeaux 2, 146 rue Leo-Saignat, 33076 Bordeaux, France.
SO Journal of physiology, (1998 Jun 15) 509 (Pt 3) 817-31.
Journal code: 0266262. ISSN: 0022-3751.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980817
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Entered Medline: 19980806
AB 1. The effects of low concentrations of domoate, an agonist at both alpha-amino-3-hydroxy-5-methylisoxazole-4-propionate and kainate receptors (AMPARs and KARs, respectively), were investigated in stellate cells in slices of mouse cerebellum at two developmental stages (postnatal day (PN) 11-13 and PN21-25). 2. Low concentrations of domoate enhanced the frequency of miniature IPSCs (mIPSCs) recorded in the presence of tetrodotoxin (TTX) at PN11-13 but not at PN21-25. 3. The effects of low concentrations of domoate on synaptic activity were probably mediated by the activation of AMPARs and not KARs, since they were blocked by GYKI 53655 (LY300168), a selective **AMPAR antagonist**. 4. Domoate increased mIPSC frequency in part by activation of presynaptic voltage-dependent Ca2+ channels since potentiation was reduced by 60 % in the presence of Cd2+. AMPARs in stellate cells were found to be permeable to Ca2+. The residual potentiation in the presence of Cd2+ could thus be due to a direct entry of Ca2+ through **AMPAR** channels. 5. In the presence of TTX, potentiation of synaptic activity by focal application of domoate was not restricted to the region of the cell body, but was observed within distances of 120 micro(m). These experiments also revealed a strong spatial correlation between the location of the presynaptic effects of domoate and the activation of postsynaptic AMPARs. 6. Our data show a developmentally regulated presynaptic potentiation of synaptic transmission between cerebellar interneurones mediated by AMPARs. We discuss the possibility that the developmental switch could be due to a shift in the localization of AMPARs from the axonal to the somato-dendritic compartment.

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